DOES NOT CIRCULATE

UNIVERSITY
OF MICHIGAN
AUG 10 1954
MEDICAL
LIBRARY

The Journal of the

International College of Surgeons

FOUNDED BY DR. MAX THOREK

Symposium

on

Neurosurgery

The Spine and Spinal Cord

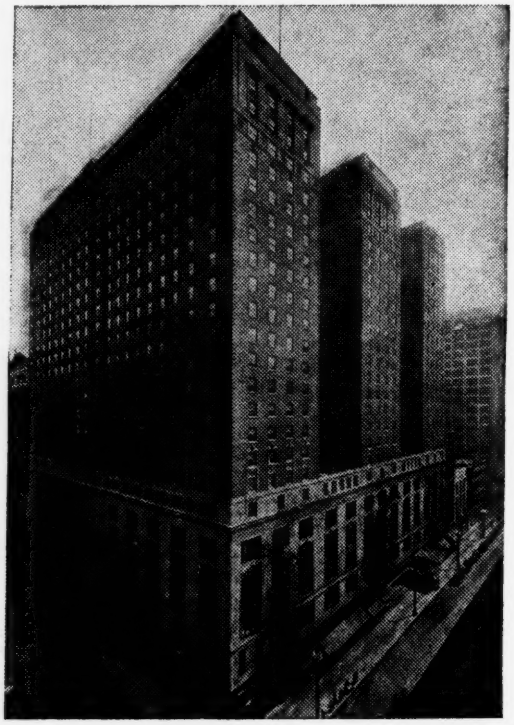
DR. JAMES WINSTON WATTS, F.A.C.S., F.I.C.S.
Guest Editor

Vol. XXII *July, 1954* No. 1

610.5
I6
C7j
V.22

PLAN TO ATTEND

THE NINETEENTH
CONGRESS AND CONVOCATION
of the
UNITED STATES AND CANADIAN SECTIONS
of the
INTERNATIONAL COLLEGE OF SURGEONS
at



PALMER HOUSE—CHICAGO
Congress Headquarters

September 7 to 10 inc., 1954

For information regarding preliminary program and reservations, etc., address:

Dr. Karl A. Meyer, F.A.C.S., F.I.C.S., Secretary
1516 Lake Shore Drive
Chicago 10, Illinois

The Journal of the International College of Surgeons

FOUNDED IN GENEVA, SWITZERLAND, 1935 - INCORPORATED IN WASHINGTON, D. C., 1940

Editor-in-Chief
MAX THOREK, M.D., Sc.D., LL.D., F.B.C.S., F.I.C.S., F.P.C.S. (Hon.), F.R.S.M.

Associate Editor
PHILIP THOREK, M.D., F.A.C.S., F.I.C.S.

Consulting Editor
MORRIS FISHBEIN, M.D., F.I.C.S. (Hon.)

Assistant Editors
Dorothy Langley and Dora Stone

Production Assistant
Frances Cretcher

Publication Committee

Francisco Graña, M.D., *Chairman*
Rudolph Nissen, M.D.
Henry W. Meyerding, M.D.
Max Thorek, M.D., *Editor-in-Chief*
Arnold S. Jackson, M.D.
Edward L. Compere, M.D.

Summary Editors

Dr. Manuel A. Manzanilla, Jr., Mexico, D.F.
Spanish
Dr. Virgilio Alves de Carvalho Pinto, São Paulo
Portuguese
Dr. Fritz Rothbart, Chicago
German
Dr. Antonello Franchini and Dr. Marino Mini, Bologna
Italian
Dr. Albert Jentzer, Geneva
French

News Editors
ASSOCIATE SECRETARIES—Various Countries

CONTENTS—JULY, 1954

SYMPOSIUM ON SPINE AND SPINAL CORD

Tumors of the Foramen Magnum 1
J. Grafton Love, M.D., F.A.C.S., F.I.C.S. (Hon.) Emil P. Thelen, M.D., and Henry W. Dodge Jr., M.D., Rochester, Minnesota

Focal Spinal Arachnoiditis Complicating Spinal Anesthesia 18
Jonathan M. Williams, M.D., F.A.C.S., Washington, D. C.

Spinal Cord Reflexes 30
Karl-Erik Hagbarth, M.D., D.A.B., Stockholm, Sweden

Spinal Echinococcosis 35
Feyyaz Berkay, M.D., F.I.C.S., Istanbul, Turkey

Physiologic Mechanisms of Spinal Shock 44
George M. Austin, M.D., and Grayson P. McCouch, M.D., D.A.B., Philadelphia, Pennsylvania

Relief of Spasticity in Paraplegia (Abstract and Discussion) 53
Irving S. Cooper, M.D., New York City, New York

Surgical Treatment of Congenital Spinal Disorders (Abstract and Discussion) 59
Donald Matson, M.D., F.A.C.S., Boston, Massachusetts

Bladder Disturbances and the Management of Patients With Injury to the Spinal Cord (Abstract and Discussion) 61
Ernest Bors, M.D., Long Beach, California

Evaluation of Skeletal Traction in the Treatment of Cervical Spine Injuries (Abstract and Discussion) 63
William Gayle Crutchfield, M.D., Charlottesville, Virginia

Experimental Regeneration in the Spinal Cord (Abstract and Discussion) 66
W. W. Chambers, Ph.D., Philadelphia, Pennsylvania

OTHER ORIGINAL ARTICLES

Tumors of the Brain and Spinal Cord as They Concern the General Surgeon 68
Hendrik J. Svien, M.D., F.A.C.S., and Andrew L. Karavitis, M.D., Rochester, Minnesota

Transabdominal Anterior Discographic Examination 76
John I. Perl, M.D., F.A.C.S., Chicago, Illinois

A New Method for Anesthetizing the Inferior Alveolar Nerve 85
Ziya Camal B. Aksoy, M.D.†, D.S., F.I.C.S.

Spontaneous Intracerebral Hematoma 89
William R. Chambers, M.D., William W. Moore, M.D., and Exum Walker, M.D., Atlanta, Georgia

Venous Angioma: A Cause of Cerebral Palsy 94
Samuel Rosner, M.D., F.I.C.S., New York City, New York

Cerebral Venous Angioma Treated by Electrocautery 98
Samuel Rosner, M.D., F.I.C.S., New York City, New York

Diastematomyelia 100
Edward Martin Gates, M.D., F.A.C.S., and James A. Morton, M.D., F.A.C.R., Pontiac, Michigan

EDITORIAL

Section on Neurosurgery: A New Field of Cooperative Endeavor 103
James Winston Watts, M.D., F.A.C.S., F.I.C.S.

NEW BOOKS 105

†Deceased

Medical

FOUNDED BY DR. MAX THOREK

The Journal of the International College of Surgeons

FOUNDED IN GENEVA, SWITZERLAND, 1935 - INCORPORATED IN WASHINGTON, D. C., 1940

Vol. XXII JULY, 1954 No. 1

Symposium

Tumors of the Foramen Magnum

J. GRAFTON LOVE, M.D., F.A.C.S., F.I.C.S. (Hon.)

EMIL P. THELEN, M.D., AND HENRY W. DODGE JR., M.D.

ROCHESTER, MINNESOTA

SINCE tumors of the foramen magnum are relatively uncommon and since headache and cervical pain (early and often initial symptoms of such tumors) are such frequent complaints of patients in general, these lesions are likely to go unrecognized until serious and often tragic neurologic sequelae have occurred.

A large number of neoplasms occurring at this level of the neuraxis are either frankly benign or of such low grade malignancy that the long-term prognosis after total removal is favorable. The prognosis for highly malignant tumors of this region varies little, of course, from that of such tumors located elsewhere in the body. However, it seems likely that the eventual surgical result can be greatly bettered in the case of benign and slowly growing malignant growths occurring at the foramen magnum if the diagnostic team manifests early suspicion and the neurosurgeon early endeavor. Also, fortified surgical optimism is a valuable adjunct.

The serious nature of tumors of the foramen magnum is attested to by the fact that many of them in the past have been recorded from postmortem examination. However, the singular experience of watching a patient progress from quadriplegia and Cheyne-Stokes respiration to eventual complete recovery following surgical removal of a meningioma of this region makes even the most dubious regard headache (particularly when suboccipital), cervical pain, peculiar peripheral weakness and paresthesias with a more critical and appraising eye.

From the Section of Neurologic Surgery, the Mayo Clinic (Drs. Love and Dodge) and the Mayo Foundation (Dr. Thelen, Fellow in Neurosurgery).

Read at the Eighteenth Annual Congress of the United States and Canadian Sections, International College of Surgeons, New York, Sept. 13-17, 1953.

The pathologic material in this paper was presented as part of Dr. Thelen's thesis. The material has been reviewed by Dr. J. W. Kernohan of the Section of Pathologic Anatomy.

Submitted for publication Oct. 19, 1953.

The purpose of this paper is to present observations made in 74 cases of tumor of the foramen magnum encountered at the Mayo Clinic through December 1952, among approximately 5,400 tumors of the brain and 1,200 tumors of the spinal cord. Figure 1 portrays the general appearance of the region under consideration. Details may be obtained from any standard textbook of anatomy.

Prior Observations.—In 1888, Osler reported a glioma extending to the foramen magnum. Other early reports were made by Sokoloff and by Spiller. The latter, in 1907, reported a tumor that extended from the fourth ventricle to the level of the sixth cervical vertebra.

Significant contributions to the problem were made by Abrahamson and Grossman from 1921 to 1923, Frazier and Spiller in 1922, Bailey in 1924, Elsberg in 1925, and Tilney and Elsberg in 1926.

In 1928 Cushing and Bailey reported 5 cases in which a vascular tumor passed through the foramen magnum. Elsberg and Strauss reviewed the data on 6 cases in which an extramedullary tumor projected into the posterior cranial fossa, and observed that in only 7 of 185 cases of tumor of the spinal cord did the growth lie both in the upper vertebral canal and in the posterior fossa. In 1932 Cushing published his extensive work on intracranial tumors, with statistics.

Symonds and Meadows in 1937 added 7 cases of tumor of the foramen magnum, in 1 of which the tumor was an aneurysm of the vertebral artery. Similarly, a vertebral aneurysm was reported by Yaskin and Alpers in 1944. In 1938 Gardner and his co-workers suggested that further study of tumors in the region of the foramen magnum would be necessary to formulate a basis for recognition of these new growths.

Cushing and Eisenhardt then published a communication in which they differentiated supraforaminal (craniospinal) from subforaminal (spinocranial) tumors. They also cited Bogorodinsky, who analyzed 1 case of his own and 22 cases from the literature under the name of craniospinal tumor.

Subsequently, in 1941, Love and Adson reported 23 cases of a lesion at the foramen magnum, and other cases were reported by List in 1943, Bennett and Fortes in 1945 and Grant in 1948. The diagnostic problem of lesions at this level in the neuraxis was reviewed in 1950 by Abbott and by Piehl, Reese and Steelman.

Symptoms.—In our group of 74 cases there were 1 aneurysm of the vertebral artery and 73 neoplasms. We have been careful to include only new growths arising in, or immediately above or immediately below and extending through, the foramen magnum.

The longest duration of symptoms was more than thirteen years, and at operation in that case the symptoms were shown to be due to a meningioma. The average duration of symptoms was somewhat more than twenty-eight months. There were 39 male and 35 female patients in the series. The ages varied from 2 to 70 years, the most common ages being in the range of 31 to 50.

Pain as an initial complaint was common. It was usually felt in the postcervical or suboccipital region, but in a few cases it was in the lumbar region and the lower extremities. Occasionally pain affected an upper extremity, and rarely pain and weakness of an extremity occurred simultaneously. An increase in discomfort was characteristically caused by attempted movements of the neck and head, and patients tended to have protective limitation of motion of the neck.

An equally common initial complaint was headache, which became increasingly severe. The headaches were predominantly occipital, but frequently were described as extending forward to the parietal and frontal areas.

Weakness of an extremity was another initial complaint. This was experienced more commonly in an upper than in a lower extremity. Other primary symptoms were nausea and vomiting, peripheral paresthesias, double vision, recurrent attacks of

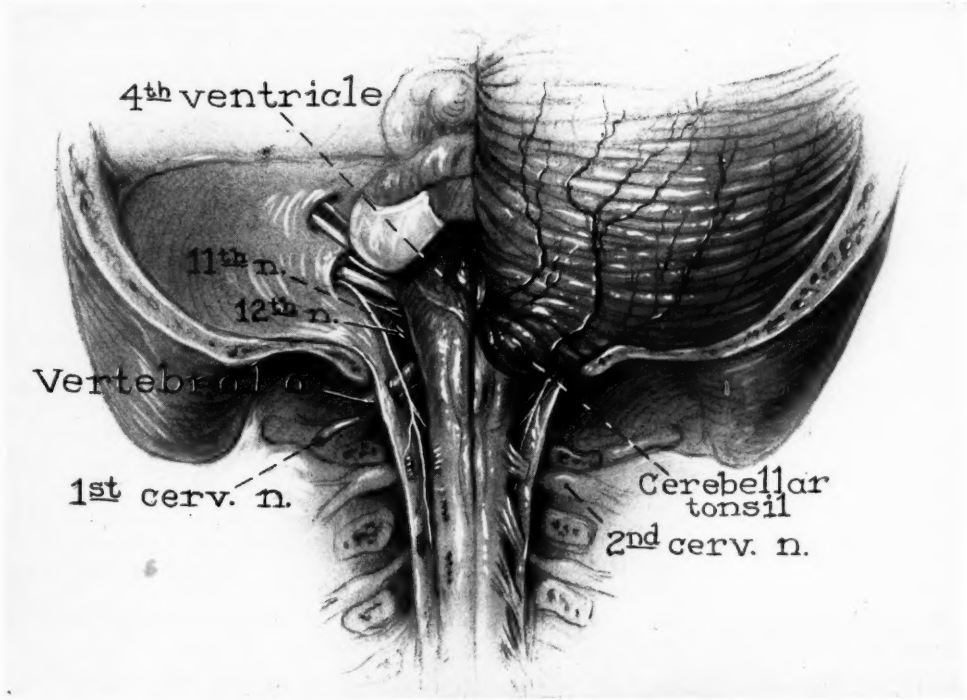


Fig. 1.—Important anatomic structures in the region of the foramen magnum.

dizziness, unsteady gait, hoarseness, and difficulty in swallowing and phonation. Several patients described a "profound weakness" (fatigue), which prevented them from doing anything.

An unusual and difficult symptom to evaluate was the early occurrence of low back pain and severe unilateral or bilateral sciatica in 2 cases. The association of low back and sciatic pain with lesions of the cervical cord has been noted by Camp.

Paresthesias (numbness, tingling, burning, itching) were present at some time in 45 cases.

There were no motor or sensory symptoms in 15 cases, while combined motor and sensory difficulties were present in 36 of the 74 cases. Sensory but no motor symptoms were recorded in 7 cases, and motor but no sensory abnormalities were observed in 9. Six persons described a feeling of generalized weakness without alteration of sensation, and 1 described profound weakness of all extremities, but the neurologist was unable objectively to show sensory deficit. In 2 of the 7 patients complaining of generalized weakness the records indicated that the attacks of weakness were transitory but repetitious.

In 49 cases there were histories of progressive motor weakness, although it was difficult to establish any particular pattern of loss of motor function. Case 1 is a good example of the problem presented in the diagnosis of these tumors, and well demonstrates the need for suspecting tumors when confronted by progressive lesions near the foramen magnum.

REPORT OF CASES

CASE 1.—A 43-year-old white married woman, who was first examined at the Mayo Clinic in June 1951, had a primary complaint of swelling and burning of the hands and a feel-

ing of weakness. Over the previous eight years she had noticed increasing pain and tingling in her hands, with a tendency for the hands to "go to sleep." In the two or three years preceding her visit to the clinic she had noticed increased subjective weakness of the hands and swelling of the fingers, and over the preceding twelve months she had felt a "constricting band" around the abdomen.

The results of general physical examination were within normal limits. Routine laboratory studies, including a blood count, urinalysis and blood chemical determinations, gave normal results. The blood sedimentation rate (Westergren method) was 53 mm. in one hour, and the blood serologic reaction for syphilis was negative. Routine roentgenograms of the cervical portion of the spinal column, chest and skull were reported as not disclosing any abnormality. Neurologic examination revealed only slightly increased deep tendon reflexes. Because of the failure to discover definite neurologic or general physical abnormalities, medical therapy was prescribed and the patient was advised, if her complaints persisted or became worse, to return for reexamination.

Fig. 2.—Myelogram showing block at the level of the first cervical vertebra and outlining the inferior portion of the meningioma described in Case 1. At times herniation of the cerebellar tonsils gives just such a picture and may be confused with a tumor at this level.

In October 1952, approximately sixteen months later, the patient was readmitted to the clinic seriously ill. She was having respiratory difficulty. The cranial nerves seemed intact on neurologic examination; the right arm and leg were paralyzed, and the left arm and leg were very weak. Signs related to the arms and legs indicated bilateral involvement of the pyramidal tracts. Superficial sensation was greatly impaired from the region of the second cervical dermatome down, although in the legs the sensation was somewhat better. Vibratory sense was impaired in the shoulders and sternum but seemed intact in the legs. Joint sense was recorded as normal except in the right hand.

Because of the patient's rapidly worsening condition, she was placed in a chest respirator. During the preliminary investigation her consciousness was intact and there was little evidence of increased intracranial pressure. She was able to state that a neurosurgeon in her home community had recently taken a myelogram, which was considered not to have shown any abnormality.

Because of the suspicion of a lesion in the high cervical region or in the foramen magnum, another myelogram was taken. This revealed a filling defect in the column of contrast medium (pantopaque) at the level of the first cervical vertebra (Fig. 2), and immediate operation was advised. Tracheotomy was performed as a preliminary measure. Then combined suboccipital craniectomy and cervical laminectomy were performed and a meningioma of the foramen magnum removed. This tumor was on the left anterolateral aspect of the cord and medulla.

After the operation the patient made a rapid recovery, and when dismissed, on Nov. 4, 1952, she was able to walk out of the hospital. She was seen again after dismissal, and her improvement was continuing.

Neurologic Data. — In 38 cases there were no objective sensory disturbances, and 4 additional patients could not be adequately examined for these changes. Three patients complained of loss of pain and temperature sensation on a part of one side of the body and loss of touch sensation on a part of the opposite side, yet none exhibited the typical features of the Brown-Sequard syndrome. In 7 cases the

area of loss of pain and temperature sensation coincided with the deficit to touch. In 2 cases the loss was on the same side but in different areas.

An objective loss of pain sensation was present in 32 cases. Loss of temperature appreciation paralleled the loss of pain except in 3 instances. In these there was a definite loss of the sense of pain, but temperature appreciation was normal.

Tactile sensation was definitely absent in 17 cases. Loss of vibratory sense was recorded in 24 cases, and a decided reduction or loss of joint sense was noted in 19 cases. In general, loss of joint awareness and vibratory sense paralleled the loss of tactile discrimination.

Astereognosis was reported in 18 cases, in 12 of which it was bilateral. Numerous authors have criticized the use of the term "astereognosis" whenever there is an alteration of sensation. In our group of patients, sensation was disturbed in 16 of the 18 cases.

Coordination was impaired in 56 cases, and the Romberg sign was positive in 28 cases. Disturbance of gait occurred in 63 cases, in 12 of which the patients were unable to walk.

Impairment of strength was encountered in 49 persons, 6 of whom exhibited hemiparesis. There was involvement of all four extremities in 17 cases. The upper extremities alone were involved in 5 cases, in 1 of which the weakness was confined to the shoulder girdle. In 4 cases both upper extremities were affected. In 3 cases there was weakness of both legs, and in 1 case there was involvement of only one leg. In 7 other cases no weakness was reported except that due to dysfunction of the cranial nerves. In 3 instances the strength could not be adequately tested.

Muscular atrophy was recorded in 17 cases. In 12 of these there was atrophy of the small muscles of the hands, and in 7 instances this was bilateral. In the remaining cases the atrophy was located in the shoulder or arm muscles. Fibrillation occurred in 11 cases, in 3 of which it was noticed in the tongue, and in the remainder of which it occurred in the upper extremities.

Reflex changes were present in 64 cases. An increase in the tendon reflexes of one or both sides was the common observation. In 8 cases the corneal reflex was diminished or absent, being affected bilaterally in 6. There was involvement of the palate or pharyngeal reflexes in 10 cases, in 7 of which it was bilateral. There was involvement of one or more of the cranial nerves in 46 cases.

Papilledema was recorded in 37 cases. It was not noted in cases of the spinocranial tumor, but it has been observed in cases of tumor of the cervical cord.[17] In the cases in which blindness was present there was a high degree of choke, with evidence of optic atrophy. In 18 cases there was paresis of one or more of the extrinsic muscles of the eye. In the few patients with changes in the visual fields, the defect usually could be accounted for on the basis of hydrocephalus of the third ventricle or a high degree of choke.

There was involvement of the region supplied by the trigeminal nerve, as evidenced by diminished corneal reflexes or loss of facial sensation, in 15 cases; in 2 additional cases there was also weakness of the muscles of mastication. There was involvement of the facial nerve in 9 cases, in 6 of which it was of the peripheral type. Hearing was diminished in 8 cases. There was evidence of involvement of the glossopharyngeal and vagus nerves in 15 cases. The spinal accessory nerve was affected in 14 cases, the condition being bilateral in 9. The hypoglossal nerve was involved in 10 cases. Although Horner's syndrome in complete form was not present in any of these cases, there was evidence of impairment of the sympathetic nervous system. In 10 cases miosis was noted. In 1 case there was absence of sweating on one side of the face. Nystagmus was present in 41 cases.

Electroencephalographic study has not proved of much aid in our hands in the diagnosis of tumors at this level of the neuraxis, except to arouse further sus-

Fig. 3.—Degenerating cystic neurofibroma measuring 6 by 3 by 2 cm. and weighing 7.8 Gm., which extended from the third cervical level up to and through the foramen magnum. This tumor was removed from a 37-year-old white man who had had unexplained early morning cervical pain for two years before suddenly experiencing peculiar paresthesias and weakness of the arms and legs two weeks prior to admission to the clinic.

picion in the occasional case of a lesion that is situated in the posterior fossa and that may be associated with bilateral occipital or posterior parietal dysrhythmias.

Until recently electromyographic methods have not been employed, but it does seem likely that, if judiciously applied, this mechanical test might at times give valuable clues as to the disease process comprising the upper part of the cervical portion of the spinal cord. This may have been true in the following case:

CASE 2.—A 50-year-old white married physician, who was first examined at the Mayo Clinic in January 1950, had a chief complaint of muscle spasms of the neck of one year's duration. He stated that approximately eleven months before admission he had been straining at stool and had experienced a sharp pain over the lower posterior portion of his neck. This pain was intermittent, but became more frequent and did not respond to any particular treatment. Over the next several months the pain was accompanied by spasm of the cervical muscles and soreness, and the patient became increasingly nervous and experienced considerable fatigue. He was unable to obtain relief from spasm of the cervical muscles and the cervical pain by any of the usual methods. It had been intimated strongly to him elsewhere that his pain was on a functional basis.

The results of general physical examination, laboratory tests and roentgenographic examination of the skull, the cervical portion of the spinal column and the chest were all within normal limits. The blood serologic reaction for syphilis was negative. Urinalysis gave negative results. The observations on careful neurologic examination were within normal limits except for slight horizontal nystagmus on lateral gaze and occasional double vision on downward gaze. Electroencephalographic investigation, examination of the fundus and studies of the visual fields were all interpreted as giving results within normal limits.

Because of the persistent suboccipital and posterior cervical discomfort and the slight nystagmus with occasional diplopia, a lesion of the posterior fossa or the foramen magnum was suspected. Examination of the spinal fluid, which was made without a Queckenstedt test, revealed a protein content of 75 mg. per hundred cubic centimeters and a lymphocyte count of 2 per cubic millimeter. The gold sol curve was normal, and the Kolmer reaction for syphilis was negative.

At exploration carried out on February 1, a large Grade 2 astrocytoma was encountered. This passed from the posterior fossa down through the foramen magnum to the upper part of the cervical portion of the spinal cord.

After the operation the patient made a satisfactory recovery. When he was last examined, in June 1953, careful neurologic investigation failed to reveal evidence of recurrence.

Roentgenologic Aspects. — Routine roentgenographic examination of the skull and the cervical portion of the spinal column was of aid in 32 of the 74 cases. In 2 cases neither the roentgenograms nor the reports on them were available. The most common abnormalities, listed in descending order of frequency with which they were encountered on routine roentgenographic survey, are given in Table 1.

At the Mayo Clinic, routine roentgenograms of the skull include postero-anterior, stereoscopic, lateral and Towne views. Anteroposterior, lateral and usually three-quarter views of the cervical portion of the spinal column also are obtained when a cervical lesion or a tumor at the foramen magnum is suspected.

Contrast radiologic studies were done in 14 cases. A pneumoencephalogram made in 1 case revealed an anterior displacement of the aqueduct. Ventriculograms were taken in 7 cases as a diagnostic procedure; in 6 of these cases a diagnosis of a lesion in the posterior fossa was made, while in the other, internal hydrocephalus was the only diagnosis that could be made. Six

patients underwent myelographic studies, 2 with lipiodol and 4 with pantopaque. In 1 case myelographic study gave negative results, while in the others it revealed an obstruction at the appropriate site. One patient had had contrast studies elsewhere, but the results were not available to us.

Cerebrospinal Fluid.—The cerebrospinal fluid was examined in 27 cases. Complete block was present in 6 cases and a partial block in 4 in which the Queckenstedt test was made. Often, when we suspect a neoplasm in the posterior cranial fossa or at the foramen magnum, if there is no edema of the optic discs we do a diagnostic lumbar puncture to obtain fluid for protein determination without doing a Queckenstedt test.

The protein content of the fluid was increased in 22 cases. In 12 cases it was more than 100 mg. per hundred cubic centimeters, ranging between 110 and 4,000 mg., while in 4 cases it was between 70 and 90 mg. In the remaining cases the protein level was 60 mg. or less, being 20 mg. in 2 cases.

In 3 cases the neurologic symptoms and signs were greatly increased after spinal puncture. In 2 cases an exacerbation of symptoms followed a spinal puncture at home.

Surgical Aspects.—To the best of our knowledge, curing a patient harboring a tumor of the nervous system is best effected by complete removal of that tumor, benign or malignant. Surgical intervention may be complemented at times by high voltage roentgen or radium therapy. Occasionally it is deemed necessary to resort to primary irradiation without surgical intervention, in the hope of palliation. The benefit of this is often only temporary.

The removal of a tumor originating within the posterior cranial fossa and extending downward through the foramen magnum into the cervical spinal canal (craniospinal), or a tumor arising primarily in the cervical spinal canal and growing upward (Fig. 3) through the foramen magnum (spinocranial) is ordinarily accomplished by means of a combined cervico-occipital operation, that is, a suboccipital craniectomy is performed and extended to include an upper cervical laminectomy.

Such an operation is best performed with the patient in the upright position, through a midline incision extending from a point 3.5 cm. above the external occipital protuberance downward across the tips of the bifid cervical spinous processes. The length of the downward extension depends on the extent of the intraspinal portion of the tumor, and this often cannot be determined until the lower portion of the neoplasm has been exposed. Whenever possible, laminectomy should be of the unilateral or hemilaminectomy type, so that the normal stability and flexibility of the cervical part of the spinal column may be preserved. At times, because of the size or position of the tumor, it is essential to remove both laminae of the cervical vertebrae to secure adequate exposure. In any event, reflection of the muscles should be subperiosteal, the articulating facets should be preserved, and accurate suturing of the different anatomic layers should be carefully attended to in closure of the wound. Hemostasis must be meticulous, and observance of the strictest aseptic technic is essential (Fig. 4).

With patients evidencing weakness of the pharyngeal and laryngeal muscles we are relucant to use general anesthesia and have found combined sedation and local infiltration of the operative site extremely

TABLE 1. — *Roentgenographic Data Associated with Tumors of the Foramen Magnum and Listed in Descending Order of Frequency of Occurrence in 74 Cases*

1.	Evidence of increased intracranial pressure
	a. Erosion of clinoid processes
	b. Separation of sutures
	c. Increased convolutional markings
2.	Widening of interpedicular spaces of the upper cervical vertebrae
3.	Enlargement of the foramen magnum, with or without evidence of bony erosion
4.	Calcification in or about the foramen magnum
5.	Enlargement of an upper cervical intervertebral foramen

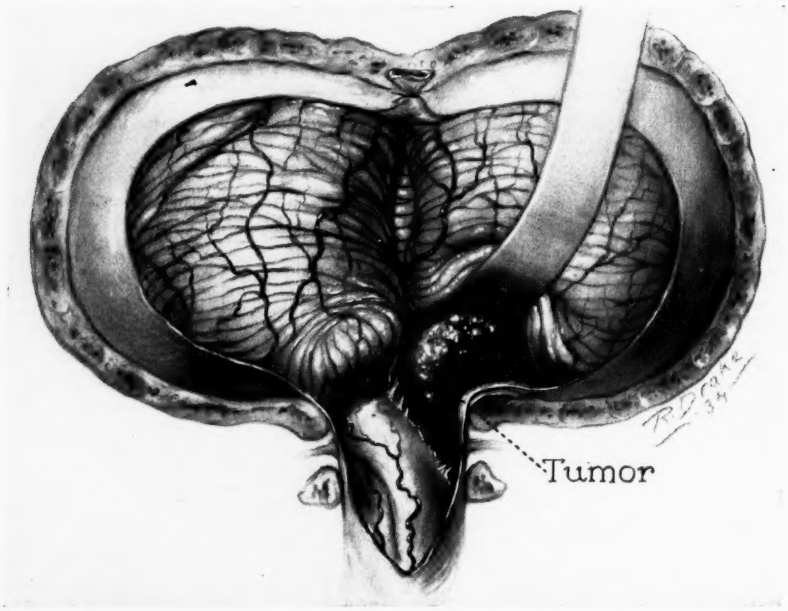


Fig. 4.—Surgical exposure of an endothelioma situated posterior to the upper cervical portion of the spinal cord and extending upward (spinocranial) through the foramen magnum.

useful and, in our opinion life-saving. The majority of patients in the past have been operated on with endotracheal open drop ether anesthesia. More recently, combined pentothal-nitrous oxide-oxygen-curare anesthesia has been used.

Surgically the greatest advance in the successful handling of patients with compressive lesions of the upper cervical part of the cord and the lower part of the brain stem seems to us to be the judicious use of preoperative or early postoperative tracheotomy to permit an unobstructed airway, better oxygenation of the blood, and better aspiration of accumulated secretion in those cases in which respiratory stimulus or muscle power is inadequate. In such cases, the use of antibiotics, oxygen therapy, blood replacement, and maintenance of fluid and electrolyte balance are, of course, routine.

From the standpoint of surgical extirpation, separation of tumors of the foramen magnum into "supraforaminal" and "subforaminal" types is chiefly of academic interest. Except for those intramedullary neoplasms and those situated anterior to the medulla and involving the basilar groove, the difficulties of surgical extirpation are about the same with respect to both the aforementioned classifications. Any operative procedure at this level involves grave danger, because of the proximity of so many vital centers in the medulla oblongata (Fig. 5).

The surgical mortality rate associated with tumors of the foramen magnum is likely to be high, for many cases are encountered by the neurosurgeon only when late signs of medullary encroachment are in evidence. Of the 74 patients in our series, 34 died during the postoperative period (at the Mayo Clinic any patient who dies after an operation while he is still hospitalized is considered statistically to represent a postoperative death, regardless of the time interval between surgical intervention and death).

Death most commonly was the result of respiratory failure. It was rarely caused by infection, uncontrolled hemorrhage or postoperative shock. The confusing surgical diagnostic problem that may be presented by tumors at this level, and which is well illustrated by Case 3, is undoubtedly the cause of late recognition and late surgical therapy in many instances.

CASE 3.—A 42-year-old white married woman, who was first examined at the Mayo Clinic in June 1950, presented the main complaints of double vision, nausea and vomiting, and cervical pain. A carefully taken history revealed that approximately six months earlier, after much strenuous activity, she had begun to have effortless vomiting. This persisted, and she gradually became weaker. Because of the persistent vomiting the diagnosis of gallbladder disease was made elsewhere and the gallbladder was removed, as well as the appendix.

In March 1950, during the hospitalization for this operation at another clinic, the patient began to notice some loss of visual acuity, and complained so much of pain in the neck and difficulty with the bowels that she was given a course of electroshock therapy. Three months prior to admission to the Mayo Clinic she had returned home and had begun to experience dizzy spells with double vision. The nausea and vomiting had continued, and the patient was considerably weakened. Because of her continuing complaints and her refusal to attempt to walk, she was given another shock treatment.

Since her complaints of posterior cervical pain, dizziness, vomiting and weakness were increasing, the patient was brought to the clinic. Neurologic examination revealed much splinting of the head and neck and grimacing when an attempt was made to move the head passively. There was palsy of the right external rectus, and the patient exhibited a bizarre gait, which was seemingly functional. She talked incessantly of multiple functional complaints. The roentgenographic appearance of the skull, the cervical part of the spinal column and the chest was within normal limits. There was edema of the right optic disc, of 1 diopter. The left optic nerve head was considered normal. Visual field studies showed a left inferior nasal and relative inferior altitudinal anopsia with central scotoma and an enlarged blind spot on the right—features characteristic of the syndrome of a left basofrontal lesion. The electroencephalographic

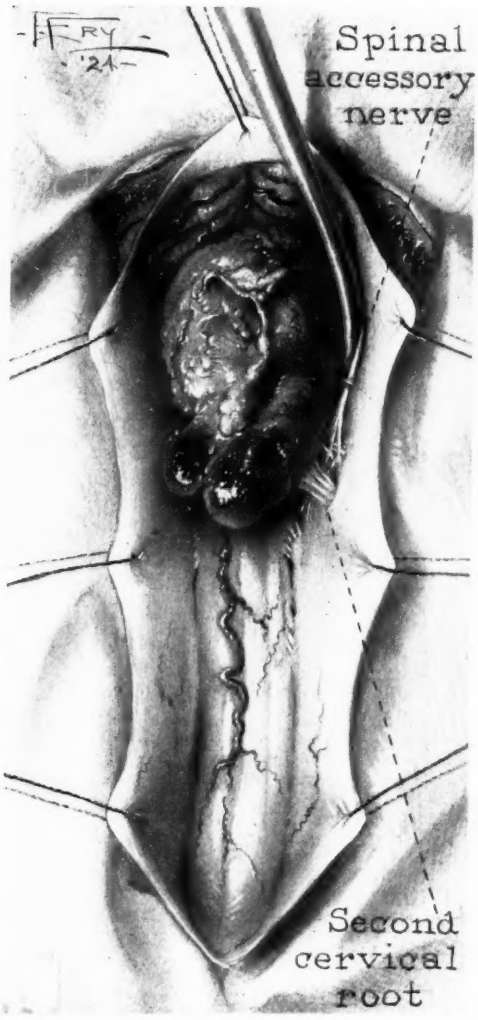


Fig. 5.—Surgical exposure of a chordoma within the foramen magnum that was causing marked compression and distortion of the medulla oblongata. (*Reproduced by permission from Adson, A. W.; Kernohan, J. W. and Woltman, H. W.: Cranial and Cervical Chordomas: a Clinical and Histologic Study, Arch. Neurol. & Psychiat. 33: 247-261 [Feb.] 1935*).

patterns were interpreted as normal. The patient was suspected of having an indeterminate intracranial lesion, and a ventriculogram was made. This revealed a minimal amount of symmetric internal hydrocephalus and good filling of the entire ventricular system, includ-

ing the fourth ventricle. It was decided to explore the foramen magnum and the upper cervical part of the cord. At operation a large metastatic Grade 2 adenocarcinoma (hypernephroma type) was observed, extending from the right cerebellar lobe through the foramen magnum into the cervical part of the spinal cord. Convalescence from the operation was uneventful, and the patient was dismissed home on the ninth postoperative day.

Pathologic Characteristics. — As has been indicated, of the 74 lesions causing compression of the neuraxis at the foramen magnum, 73 were neoplasms and 1 was an aneurysm of the vertebral artery. The verified histologic diagnoses are given in Table 2. Nineteen of the lesions were of the spinocranial type, arising from below the foramen magnum and projecting cephalad through it, and 54 were of the craniospinal type, that is, the tumors originated cephalad to the foramen and projected down through it. The remaining case was that of the aneurysm of the vertebral artery.

It is of great importance and interest to note the large number of meningiomas and low-grade malignant lesions encountered (Table 2). There was excellent correlation between the type of tumor encountered and the duration of symptoms. The correlation between the age of patient and the type of lesion is shown in Table 2. Because of the benignancy or low-grade malignancy of many of these tumors, accurate, early diagnosis is imperative if better results are to be obtained.

COMMENT

Critical evaluation of our 74 cases and review of those reported in the literature led us to conclude that no characteristic syndrome can be ascribed to operable tumors of the foramen magnum. When the patient comes for diagnosis and treatment

TABLE 2.—*Distribution of Cases of Tumor of the Foramen Magnum According to Age of Patient and Type of Tumor*

Type of Tumor	Age, Years 0-10	11-20	21-30	31-40	41-50	51-60	61-70	Total Cases
Meningioma	—	—	2	4	11	2	2	21
Astrocytoma	4	2	4	1	3	2	—	16
Ependymoma	6	2	2	3	1	—	—	14
Hemangioendothelioma	—	—	1	1	2	—	1	5
Medulloblastoma	4	—	—	—	—	—	—	4
Neurofibroma	—	2	—	1	—	—	—	3
Oligodendroglioma	—	1	—	—	1	—	—	2
Chordoma	1	—	—	—	—	1	—	2
Melanoepithelioma	—	—	—	1	—	1	—	2
Hemangioblastoma	—	—	—	1	—	—	—	1
Chondrosarcoma	—	—	—	—	—	1	—	1
Intramedullary glioma (type?)	—	—	—	—	—	—	1	1
Metastatic adenocarcinoma (hypernephroma type)	—	—	—	—	1	—	—	1
Aneurysm of vertebral artery	—	—	—	1	—	—	—	1
Total cases	15	7	9	13	19	7	4	74

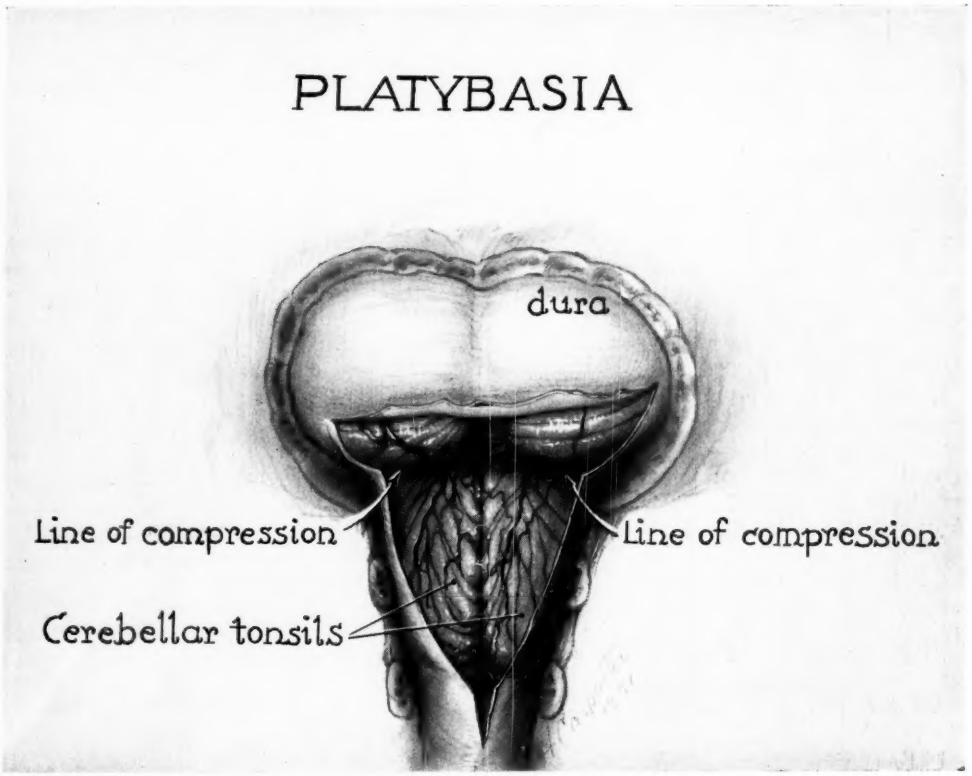


Fig. 6.—Platybasia which had produced symptoms similar to those of a tumor of the foramen magnum.

with the full-blown picture of compression by a tumor at the foramen magnum, his lesion is likely to be inoperable and incurable and to be seen first by the pathologist rather than by the surgeon.

It is evident, however, that despite the bizarre variation of symptoms and neurologic signs and the marked variation in the history of progression of the disorder, there are some factors that are relatively common and should raise the examiner's index of suspicion of a lesion at this level. Although many of the patients harboring these tumors, because of unusual symptoms and neurologic patterns are early suspected of functional difficulties, steady, relentless progression of the offending lesions is almost universal.

Pain in the upper cervical region, aggravated by motion of the neck, is most common. This, in association with suboccipital or occipital headache, occurred in the majority of our cases.

Weakness and paresthesias, usually involving the upper extremities primarily, were prominent in most cases, and most typically there was progression to involve the lower extremities. The area supplied by the second cervical nerve was affected in somewhat more than a fifth of the cases.

Vestibular and cerebellar dysfunction, as manifested by nystagmus, disturbances of gait and station and lack of co-ordination, occurred frequently. However, the pattern of motor loss was variable.

Muscular atrophy and fibrillation occurring in neural segments not directly involved by tumor suggest that some of the

bizarre symptoms may have vascular compression as a partial basis.

The type of tumor causing pressure seems to be a definite factor in determining the symptoms and signs encountered. Thus, tumors arising in the region of the fourth ventricle or cerebellum (medulloblastomas, astrocytomas, ependymomas, hemangioendotheliomas, etc.), passing through the foramen magnum and accompanied by signs of increased intracranial pressure, do not present the diagnostic problem exhibited by tumors arising at the foramen which do not cause ventricular obstruction.

It seems that there is no single method or test by which the examiner can unerringly ferret out these new growths in and about the foramen magnum. There are, however, several approaches which, if judiciously applied, will give satisfactory information.

Of prime importance is the detailed, exact chronologic history obtained from the patient. Next, careful general physical and neurologic examinations, with indicated laboratory tests and roentgenograms of the chest, the skull and the cervical part of the spinal column, are required.

If the patient shows signs of increased intracranial pressure, choked disc, etc., with cerebellar or vestibular involvement, then the diagnostic procedures indicated are those typically employed by the neurosurgeon for lesions of the posterior fossa, plus exploration with or without preliminary ventriculographic study. Should the patient manifest cerebellar vestibular dysfunction or dysfunction of the upper cervical part of the spinal cord without evidence of obstruction to the ventricular pathways, the problem becomes more difficult.

In differential diagnosis the examiner should consider tumor, aneurysm or vascular anomaly; demyelinizing disease (multiple sclerosis); encephalitis; platybasia (Fig. 6), Arnold-Chiari malformation, a congenital anomaly (Fig. 7); high cervical disc protrusion; osteomyelitis; subluxation of upper cervical vertebrae, and syphilis, among other possibilities.

Routine roentgenograms of the skull and the cervical part of the spinal column may not reveal abnormality in the region of the foramen magnum, and therefore additional views will be required. In our opinion the most information about this region may be obtained without resorting to contrast myelograms, through stereoscopic views of the base of the skull and through platybasia and atlas-axis roentgenograms. Occasionally, Stenvers views may show the first and second cervical vertebrae obliquely and to great advantage. For a specific area in this region, tomographic study may be worth while.

A further step in the diagnostic evalution may include spinal puncture and examination of the cerebrospinal fluid, with or without the Queckenstedt maneuver. In those cases in which there is even minimal evidence of respiratory difficulty or the slightest suspicion of increased intracranial pressure (even in the absence of choking of the optic discs), the Queckenstedt test is not performed. The pneumoencephalographic method is uncommonly used by us for suspected lesions in the region of the posterior fossa or the foramen magnum. If the protein content of the spinal fluid is 100 mg. or more per hundred cubic centimeters and if there is a suggestive progressive history, it is our opinion that direct surgical exploration is indicated. The presence of partial or complete block of the spinal fluid pathways makes the diagnosis of tumor more likely. We are convinced that it is hazardous to do a cisternal puncture when the patient is considered to have a lesion about the foramen magnum.

A further adjunct to the diagnosis is the contrast myelogram, taken with either pantopaque (ethyliodophenylundecylate) or lipiodal (iodized poppy seed oil). Some criticism has been directed toward the use of iodized oil in the region of the upper cervical part of the spinal cord because of the danger of passage of some globules of oil into the basal cisternae of the head. In our opinion, it should be used if the examining team considers a contrast myelogram necessary for accurate diagnosis.

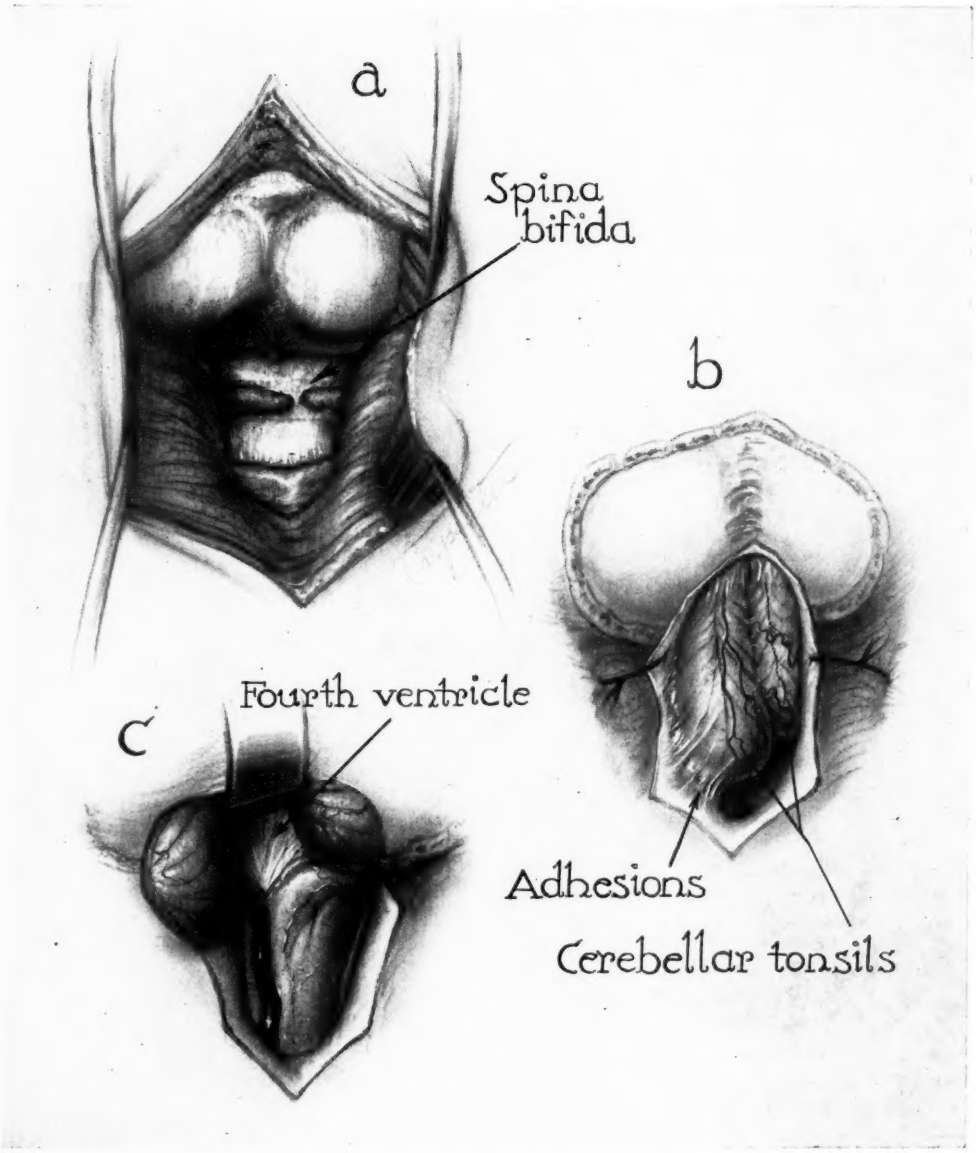


Fig. 7.—Congenital anomalies at foramen magnum may give rise to symptoms suggestive of a tumor.

It is not uncommon for the tumor to be localized clinically several segments below its actual site, and it would be unfortunate to laminectomize the cervical part of the spinal column without reward, only at a later date to recognize and remove a tu-

mor of the foramen magnum. Vertebral angiographic study can be employed as an alternate closed method of diagnosis.

Occasionally, when all examinations and tests have failed to disclose any positive evidence of a tumor and when all that remains is the diagnostic team's suspicion, direct exploration should be carried out provided the suspicion is strong enough. The patient and his relatives must first, however, be thoroughly acquainted with the advantages and disadvantages and with the comparative risks. Under such circumstances, the chance that exploration will disclose the absence of a tumor is preferable to the chance that ravages of a missed expanding lesion may be allowed to progress. Small anterior foraminal or basilar groove tumors may present just such a perplexing problem. Uncovering a degenerative lesion has little effect on the ultimate course except for the immediate surgical morbidity. Uncovering a tumor subject to palliation or cure may have a pronounced effect on the subsequent course.

SUMMARY

The authors present the Mayo Clinic series of tumors of the foramen magnum encountered through December 1952. In this group are 1 aneurysm of the vertebral artery and 73 neoplasms. The pertinent literature is reviewed.

On the basis of their experience the authors are convinced that there is no typical syndrome for the accurate clinical diagnosis of these lesions. The most frequently occurring and significant symptoms and neurologic signs are reviewed, and the aid afforded by thorough roentgen ray survey, cerebrospinal fluid examination and neurosurgical investigation are appraised.

The surgical and surgical pathologic aspects of these tumors are discussed briefly, and what the authors consider a logical method of approach in dealing with them is presented. Maintenance of a high index of suspicion and early attempts at diagnosis are stressed for patients complaining of cervical and suboccipital pain, peculiar peripheral paresthesias and weakness in cases in which the history indicates that the difficulty is progressive.

DISCUSSION BY DR. BYRON STOOKEY, NEW YORK

DR. STOOKEY: The clinical signs indicative of an extramedullary tumor at the level of the foramen magnum depend on the precise origin in relation to the spinal neuraxis, on whether the tumor is ventral, lateral or dorsal, and on the exact level of origin. To gain any concept of the neurologic signs the relation of the growth to the various pathways and emerging nerves is essential in developing a syndrome. Of importance also is the nature of the neoplasm, whether soft or hard, slow or rapid in its development.

With the wealth of material in Dr. Love's paper, I hope he will subdivide these neoplasms into extramedullary and intramedullary and make for each group a clinical study of the symptoms and signs, since only by maintaining sharply defined pathologic entities can clinical signs of their presence be determined and early recognition of the symptoms be made. As has been pointed out by Dr. Love, early recognition is essential if successful removal is to be accomplished.

I note that there were 21 meningiomas and 3 neurofibromas. Thus, apparently one-third of all growths were extramedullary neoplasms. These should lend themselves to a clinical study of great diagnostic value. There are approximately twenty relatively common infiltrating tumors, namely the astrocytomas and medulloblastomas. Fourteen ependymomas are described. I presume that these arose in the fourth ventricle and extended into the cervical region and perhaps might therefore be studied profitably as a separate group.

The primary value and interest in these cases is inherent not only in the technical surgical problems they present in relation to the medulla and its vital centers, but in the diagnostic problem, so that early diagnosis can be made and surgical intervention accomplished while the growth still can be removed with minimal danger to the patient.

Surprise has been manifested that cranial nerve signs are infrequent; yet these do occur (and, in my experience, only when the tumor is ventral or ventrolateral in relation to the spinal axis) most commonly involving the eleventh and twelfth cranial nerves. The

eleventh nerve is most frequently involved, since it takes a lateral course and extends over three or four segments within the cervical canal. Weakness on complete elevation of the arm is the most most characteristic sign, the arm being only partially elevated and not to a complete right angle. This weakness may easily be overlooked unless this sign is borne in mind.

In considering the signs produced by extramedullary tumors in this region, it is well to recall that actually the uppermost segments of the spinal cord and that part of the central axis within the foramen magnum lies actually in a large basal cistern surrounded by a large amount of spinal fluid, within whose confines considerable growth may take place without compromising the neural structures, either the cranial nerves nor the long fiber tracts of the neuraxis. Consequently, tumors in the region of the foramen magnum may be relatively silent until they have attained considerable size, sufficient to cause pressure upon either the neuraxis or the cervical or cranial nerves. Infrequency of signs referrable to the cranial nerves is due not only to the relative size of the cisterna magna but to the relative infrequency of tumors primarily ventrally placed.

When a tumor is ventrally placed, pressure may be exerted upon the ventral spinal artery and cause signs that may be and have been confusing, namely, atrophy of the muscles of the upper extremity and hand whose ventral roots are not compressed and arise a number of segments lower. It seems likely that compression of the anterior spinal artery may interfere with the blood supply to the ventral horn cells a few segments lower than the point of pressure and consequently give rise to atrophy in segments distal to the point of pressure. It is to be remembered that the anterior spinal artery is formed by two branches from the vertebral artery on the ventral surface of the medulla, from which it receives its major blood supply, and descends along the ventral aspect of the spinal cord. Consequently, in the high cervical region it constitutes the main arterial supply to the ventral part of the cervical cord. Interference with its circulation would deprive the ventral horn cells of their blood supply; hence the atrophy in lower segments. Recognition of the possibility of this mechanism is important, since focal atrophy in the lower segments may be interpreted as indicative of a lesion a number of segments lower.

I am glad that Dr. Love does not use the term astereognosis when there is disturbance of primary sensation, for only confusion can result from so doing. When primary sensation is implicated one cannot use the term astereognosis, and in tumors of this region pressure upon the dorsal column interferes with primary sensation and consequently the term astereognosis should not be used.

From a surgical standpoint, whenever possible the growth should be removed by Alfred Taylor's hemilaminectomy, since this best maintains the stability of this vital area in the vertebral column. The largest cervical cord tumor I have ever resected was removed through a hemilaminectomy without damage to the spinal cord. This approach is particularly favorable for a tumor ventrally placed. If the exposure is made through a hemilaminectomy and it is found that additional space is required, it is easy to enlarge the opening at the precise area needed and thus to maintain as much of the normal structure of the neck as possible, which cannot be done satisfactorily by a complete bilateral laminectomy.

Because of the probability of respiratory difficulty, endotracheal anesthesia is desirable in many instances. The upright posture and local anesthesia I have found to be of great value, not only in facilitating respiration but in diminishing bleeding.

Dr. Love and his co-workers are to be congratulated on their accomplishments in this field, where it is obvious that they have combined their great technical skill with excellent judgment.

CLOSING REMARKS BY DR. LOVE

DR. LOVE: Apparently, the question of the importance of the second cervical nerve in the diagnosis of tumors of the foramen magnum was not emphasized as much as it should have been. Tumors arising high in the cervical region and growing upward through the foramen magnum and tumors growing downward through the foramen magnum into the upper cervical canal are very likely to press on and irritate the second cervical nerve.

As was indicated in the presentation, one of the chief symptoms, and one of the symptoms most commonly present in the series of cases reported, was pain in the suboccipital area on the side from which the tumor had taken its origin. It is important to do an unusually careful neurologic examination of patients suspected of having a lesion of the

foramen magnum. The examiner should carefully check for diminution or loss of sensation in the distribution of the second cervical nerve, for such a sensory disturbance may be the only objective evidence that can be obtained in some of these cases.

The other question was, "How many explorations were made with negative results?" Offhand, I can't remember having explored the area without finding a pathologic process, but my colleagues and I have limited surgical exploration to those patients whose condition, according to the history, has progressed, and has not responded to an adequate plan of conservative treatment. Usually, one or more definite abnormalities have been present, as in the case of the physician to whom I referred; in that instance, there was an increased total protein content in the cerebrospinal fluid. That, in association with the progression of disability to the point where the patient had been unable to carry on his practice, and the fact that an enforced vacation in a very healthful climate failed to bring improvement, convinced us that we were justified in doing a diagnostic puncture. We reasoned that a lesion of some kind must have been situated either in the posterior fossa or in the upper cervical region. In view of the increased protein content of the cerebrospinal fluid, we carried out the surgical exploration that uncovered the tumor of the foramen magnum.

As to the query about differential diagnosis: the differential diagnosis in this group of cases would include, of course, consideration of platybasia, syringomyelia, syringobulbia, multiple sclerosis or other demyelinating diseases, occipital neuralgia, cervical arthritis, and posterior fossa tumors of many different types. For instance, many patients with tumors entirely within the posterior fossa will have pain in the neck and suboccipital area and may have dizziness, vomiting, and all of the symptoms that were mentioned as occurring frequently in this group of cases. Occipital neuralgia might be distinguished by local injection of the second cervical nerve, and cervical arthritis, of course, could be eliminated by the presence of hypertrophic changes. I appreciate the fact that the presence of bony changes does not establish the patient's symptoms as due to the bony changes. Consideration of many of the factors that might be associated with that area, of course, would entail an exhaustive neurologic investigation. To make such an investigation would almost cover the field of neurology, because the symptoms in question can be caused by many different types of conditions.

DR. LOVE: I sincerely appreciate Dr. Stookey's remarks. I am glad he emphasized the points which should be emphasized. It is important for all of us to hear such remarks from a man of his experience.

As to his question about cervical or cervico-occipital dislocation: In this group of cases, I can recall only 1 patient in whom dislocation subsequently did occur, with development of quadriplegia, which made reduction and then bonegraft fusion necessary. There is a very real danger of that, and I think it should be kept constantly in mind. We must preserve the stability of the cervico-occipital region. Thank you very much.

RÉSUMÉ

Les auteurs étudient les tumeurs au niveau du trou occipital au point de vue historique, clinico-neurologique, radiographique et ventriculographique. Ils examinent en outre le liquide cephalo-rachidien et exposent le traitement chirurgical. Trois cas sont rapportés en détail.

ZUSAMMENFASSUNG

Es liegt eine Erörterung der Geschwülste des Foramen magnum vor, die die Geschichte der Erkrankung, die Symptome, das neurologische Bild, röntgenologische und ventrikulographische Untersuchungen, die Beschaffenheit des Liquors, die chirurgische Behandlung und pathologische Befunde einschliesst. Drei Fälle werden ausführlich berichtet.

RESUMEN

Se discuten los tumores del foramen magnum bajo los siguientes puntos de vista histórico, sintomatológico, de imagen neurológica, roentgenológico y ventriculográfico, de examen de líquido cerebroespinal, de tratamiento quirúrgico y de hallazgos anatomopatológicos. Se comunican tres casos en detalle.

RIASSUNTO

I tumori del forame magno sono stati trattati sotto l'aspetto storico, sintomatologico, neurologico, radiologico e ventricolografico, oltre che chirurgico e anatomopatologico. Ne sono stati descritti 3 casi in dettaglio.

RESUMO

São estudados do ponto de vista historico, sintomatologico, neurologico e ventriculografico os tumôres do "foramen magnum," sendo ainda apreciado o estudo do liquido cefalo-raquiano, o tratamento cirurgico e os achados patologicos de tais casos.

São registradas com detalhes três observação es de tais tumôres.

REFERENCES

1. Abbott, K. H.: Foramen Magnum and High Cervical Cord Lesions Simulating Degenerative Disease of the Nervous System, Ohio State M. J. **46**:645-651 (July) 1950.

2. Abrahamson, I., and Grossman, M.: Tumors of the Upper Cervical Cord, J. Nerv. & Ment. Dis. **57**:342-363 (April) 1923.

3. Bailey, P.: A Study of Tumors Arising from Ependymal Cells, Arch. Neurol. & Psychiat. **11**: 1-27 (Jan.) 1924.

4. Bennett, A. E., and Fortes, A.: Meningioma Obstructing the Foramen Magnum, Arch. Neurol. & Psychiat. **53**:131-134 (Feb.) 1945.

5. Bogorodinsky, D. K.: Cited by Cushing, H., and Eisenhardt, L., p. 179.[9]

6. Camp, J. D.: Personal communication to the authors.

7. Cushing, H.: Intracranial Tumors; Notes upon a Series of Two Thousand Verified Cases with Surgical-Mortality Percentages Pertaining Thereto. Springfield, Ill.: Charles C Thomas, Publisher. 1932.

8. Cushing, H., and Bailey, P.: Tumors Arising from the Blood Vessels of the Brain: Angiomatous Malformations and Hemangioblastomas. Springfield, Ill.: Charles C Thomas, Publisher, 1928.

9. Cushing, H., and Eisenhardt, L.: Meningiomas; Their Classification, Regional Behavior, Life History, and Surgical End Results. Springfield, Ill.: Charles C Thomas, Publisher, 1938.

10. Elsberg, C. A.: Tumors of the Spinal Cord and the Symptoms of Irritation and Compression of the Spinal Cord and Nerve Roots; Pathology, Symptomatology, Diagnosis and Treatment. New York: Paul B. Hoeber, Inc., 1925.

11. Elsberg, C. A., and Strauss, I.: Tumors of the Spinal Cord which Project into the Posterior Cranial Fossa; Report of a Case in which a Growth Was Removed from the Ventral and Lateral Aspects of the Medulla Oblongata and Upper Cervical Cord, Arch. Neurol. & Psychiat. **21**:261-273 (Feb.) 1929.

12. Frazier, C. H., and Spiller, W. G.: An Analysis of Fourteen Consecutive Cases of Spinal Cord Tumor, Arch. Neurol. & Psychiat. **8**:455-498 (Nov.) 1922.

13. Gardner, W. J.; Karnosh, L. J., and McNerney, J. C.: Meningeal Tumor in the Foramen Magnum, Arch. Neurol. & Psychiat. **39**:1302-1307 (June) 1938.

14. Grant, F. C.: Surgical Experiences with Extramedullary Tumors of the Spinal Cord, Ann. Surg. **128**:679-683 (Oct.) 1948.

15. List, C. F.: Multiple Meningiomas; Removal of Four Tumors from Region of the Foramen Magnum and Upper Cervical Region of the Cord, Arch. Neurol. & Psychiat. **50**:335-341 (Sept.) 1943.

16. Love, J. G., and Adson, A. W.: Tumors of the Foramen Magnum, Tr. Am. Neurol. A. **67**:78-81, 1941.

17. Love, J. G.; Wagener, H. P., and Woltman, H. W.: Tumors of the Spinal Cord Associated with Choking of the Optic Disks, Arch. Neurol. & Psychiat. **66**:171-177 (Aug.) 1951.

18. Osler, W.: Glioma of the Medulla Oblongata, J. Nerv. & Ment. Dis. **15**:172-176 (March) 1888.

19. Piehl, M. R.; Reese, H. H., and Steelman, H. F.: Diagnostic Problem of Tumors at Foramen Magnum, Dis. Nerv. System **11**:67-76 (March) 1950.

20. Sokoloff: Cited by Osler, W.[18]

21. Spiller, W. G.: Gliomatosis of Pia and Metastasis of Glioma, J. Nerv. & Ment. Dis. **34**: 297-302 (May) 1907.

22. Symonds, C. P., and Meadows, S. P.: Compression of the Spinal Cord in the Neighborhood of the Foramen Magnum, Brain **60**:52-84 (March) 1937.

23. Tilney, F., and Elsberg, C. A.: Sensory Disturbances in Tumors of the Cervical Spinal Cord; Arrangement of the Fibers in the Sensory Pathways, Arch. Neurol. & Psychiat. **15**:444-454 (April) 1926.

24. Yaskin, H. E., and Alpers, B. J.: Aneurysm of the Vertebral Artery; Report of a Case in which the Aneurysm Simulated a Tumor of the Posterior Fossa, Arch. Neurol. & Psychiat. **51**: 271-281 (March) 1944.

Focal Spinal Arachnoiditis Complicating Spinal Anesthesia

JONATHAN M. WILLIAMS, M.D., F.A.C.S.

WASHINGTON, D. C.

IT has now been over a half century since anesthetic agents were introduced into the spinal subarachnoid space to render surgical procedures more easy. The earliest substance used, cocaine, had short popularity because of the quick recognition of its toxicity. With the creation of procaine in 1904 by Einhorn, a better and safer substance was made available for this form of anesthesia.

The early studies of Davis, Haven, Givins and Emmet,[1] and those of Lundy, Essex, and Dernohan[2] indicated that the administration of procaine and allied agents invariably produced changes in the leptomeninges and even in the parenchyma of the nervous tissues. Little if any information, however, could be gleaned concerning any prolonged effect of these agents of anesthesia from the many happy compilations of their successful use until about the mid-1930's, when the reports of Broch, Bell, and Davidson;[3] Ferguson and Watkins,[4] and Critchley and others[5] stimulated interest in the possible detriment to nervous tissue immediately apparent after the administration of spinal anesthesia. The persistence of paralysis and numbness accompanied by loss of sphincter control was sufficiently spectacular to arouse interest as to whether spinal anesthesia was as harmless as it had at first seemed. But concern with the neurologic complications following spinal anesthesia was rather desultory until 1950, when Kennedy, Effron and Perry[6] reported, at a meeting of the American Neurological Association, 12 cases of neurologic lesions following spinal anesthesia. (Prior to this, in 1945, Kennedy had anticipated himself in reporting 3 cases.[7]) This paper stimulated vigorous interest, and the protagonists and antagonists of spinal anesthesia each had their mighty champions.[8] Clinicians, reflecting on the work of previous investigators, conceded that the anesthetic agents themselves could be toxic, but suggested that some other and additional substance, inadvertently introduced, might contribute to these neurologic disasters.[9] In this regard, the most likely miscreant was the solution used to sterilize the ampules, which in some manner might soak into the vials, probably because of a defective seal, and contaminate the anesthetic. Though this theory seems implausible, it is actually a real hazard, and Coakley at the George Washington University Hospital has informed me that he finds three or four such defective ampules a year. Coloring the sterilizing solution in which the ampules are immersed, so that any invasion of the anesthetic agent can be readily detected, has been a simple expedient in reducing the chance of this type of casualty.[10] Recently, however, Searles and Nowell[11] have studied the role of these sterilizing solutions in the production of neurologic damage and have concluded that the coloring of the antiseptic solution probably gives inadequate protection and a false sense of security to the anesthetist employing spinal anesthesia, for the dilution factor of contaminants is great. Since there is little evidence that allergy plays a part in the production of the neurologic sequelae of spinal anesthesia, and since trauma apparently plays no role at all, one must inevitably

From the Department of Neurology and Neurological Surgery George Washington University School of Medicine, Wash., D. C.

Read at the Eighteenth Annual Congress of the United States and Canadian Sections, International College of Surgeons, New York, Sept. 13-17, 1953.

Submitted for publication April 24, 1954.

return to the inherent toxicity of the spinal anesthetic agent itself as the prime factor.

These complications, in general, may be divided into two categories. In the first group the changes are wrought by the offending agent upon nervous tissues themselves. Because the neurologic consequences are the result of destruction and are apt to be immediate, they are not germane to the topic under discussion. This is what is commonly referred to in the literature as the cauda equina syndrome. This phase of neurologic complications is dealt with in the papers by Kamman and Baker;[12] Ferguson and Watson,[4] and others.[13]

The second type of reaction, and one much more likely to be delayed, is the development of chronic, adhesive arachnoiditis. Hayes and Smith[14] have described its presence in a case with symptoms as early as four days following spinal anesthesia, but it is doubtful whether the neurologic symptoms were due to the arachnoiditis present, *per se*, or to the accompanying nervous tissue damage.

The pathologic character of this delayed reaction in its earliest stages is not well known. Davis and his co-workers,[1] as well as Kamsler[15] have shown that inflammation of the leptomeninges, in varying degrees, is a constant concomitant of spinal anesthesia. This is true particularly when continuous spinal anesthesia is administered via a catheter or a plastic tube. In its completely evolved form the microscopic picture is that of a dense, collagenous, fibrous cuff surrounding and constricting the spinal cord and adjacent vessels and containing, in its interstices, scattered aggregations of inflammatory cells. Figure 1 is typical of the microscopic appearance of chronic adhesive arachnoiditis.

The diagnosis of spinal arachnoiditis is rather infrequently made by neurologists and neurosurgeons, and naturally, without operative confirmation of that diagnosis, it is held suspect in many instances. However, during the year of 1949, on three occasions, patients were seen in whose cases clinical diagnoses of tumor of the spinal cord were made, only to have it discovered at operation that dense adhesive arachnoiditis was present, which frequently involved the dura as well. Shortly after observation of the third case, a patient with far advanced carcinoma of the uterus and bilateral pain and spasticity of the lower limbs was referred to the neurosurgical service for cordotomy. In the operating room my associates and I were surprised and distressed to find such dense arachnoiditis involving the upper dorsal portion of the cord that its rotation for the performance of anterolateral cordotomy was impossible. Struck by the coincidence of an allegedly rare disorder appearing four times during a relatively short period, we sought a common denominator. Our search was rewarded by several evocative coincidences:

The surgical level of the lesion was at the sixth thoracic vertebral body in all 4 cases. In 2 of the 4 cases this was demonstrated by myelogram. The sensory level invariably included the lumbar segments and the thoracic segments up to the tenth dermatome and frequently extended as high as the eighth, but never any higher.

The clinical picture of transverse myelitis was, as a rule, that of an incomplete lesion, with some scattered sensory preservation in one or more modalities below this level. The motor involvement was that of spasticity, with spontaneous and at times painful flexor spasms.

These 4 patients all had undergone abdominal operations in the immediate past (from three months to one year earlier), for which continuous spinal anesthesia was used. These operations were all protracted, i. e., about four hours, and in 2 instances the intra-abdominal operative procedures were repeated twice, each time with continuous spinal anesthesia. The anesthetic agent was invariably procaine.

These observations naturally raised the question of a causal relation between continuous spinal anesthesia and the development of delayed adhesive arachnoiditis. The

level of the lesions was always in the neighborhood of the sixth dorsal vertebra, which represents the nadir of normal dorsal kyphosis when the patient is supine. In this position, the anesthetic agent introduced into the subarachnoid space tends to pool at a higher concentration at this level than at other points. Finally, a study of the anatomic aspects of the blood supply of the spinal cord revealed that the descending anterior spinal artery, the chief nutrient channel of the cord, dwindles away in this region, forming casual anastomoses with coronal vessels from the dorsal region and diminishing twigs of an ascending anterior spinal artery arising in the neighborhood of the conus. Therefore, any mechanical compressive interference involving the whole cord would be more devastating here than elsewhere. Figure 2 shows the blood supply of the spinal cord and its greater susceptibility at the level of the sixth dorsal vertebra.

As a result of this experience, I have searched the wards and my private practice for other cases. Five more have been found. Myelographic studies were performed in 4, in 1 of which a block was revealed at the sixth thoracic level (Fig. 3) ; 3 others showed the telltale streaking of the iodized oil at the sixth dorsal level, and in the fourth a clinical diagnosis only was made. In all respects these cases resembled the earlier ones in which operation had been performed. I should like to report what I consider a typical case of chronic adhesive arachnoiditis as a delayed sequel to the administration of continuous spinal anesthesia.

L. D., a 50-year-old Negro teacher, was first admitted to the Freedman's Hospital on June 27, 1948, with a diagnosis of intestinal obstruction. A laparotomy was done, with lysis of adhesions, subtotal hysterectomy and appendectomy. Spinal anesthesia was employed, 150 mg. of procaine being used. The patient was discharged two weeks later. On August 25 she was readmitted with a history of abdominal pain and vomiting of two days' duration. Roentgenograms indicated obstruction of the large bowel. On the following day a cecostomy was done, again with spinal anesthesia (amount of procaine not known). One week later, with the patient under continuous spinal anesthesia, a resection of the splenic flexure of the colon was done for carcinoma, 650 mg. of procaine being used. The postoperative course was not remarkable, and the patient was discharged four weeks later. Three months after her discharge she was again readmitted, with a complaint of weakness in the legs and involuntary mass movements of both legs. The condition appeared to be progressive. There was no pain. Significant observations included anesthesia of the right leg over the fourth and fifth lumbar levels and hypesthesia at the eighth thoracic dermatome, below which hypesthesia to pinprick was present. Tendon reflexes were absent in the legs, and bilateral Babinski signs were present. There was some retention of urine. The lumbar puncture yielded only a few drops of xanthrochromic fluid which promptly clotted in the tube. There was no rise on jugular compression. Roentgenograms of the bony spine were normal. Laminectomy, done shortly thereafter, revealed dense, adhesive arachnoiditis from the sixth thoracic level down. Postoperatively, complete spinal paralysis developed from the eighth dorsal level downward, and the patient failed progressively, dying in February 1949. Permission for autopsy was not granted.

In this case an intimate relation between the development of the paraplegia and the repeated administration of a spinal anesthetic seems quite evident. More frequent, however, is the appearance of the insidious onset of weakness in the legs, vague thoracic sensory levels and sphincter disturbances apparently arising spontaneously. Because of my experience with similar cases, I regularly examine the abdominal wall for scars or question the patient very closely with regard to any prior surgical treatment and the kind of anesthesia employed when tumor of the spinal cord is suspected and the clinical localization appears to be in the neighborhood of the sixth thoracic vertebra. This is a rewarding measure; it has saved at least 3 of my subsequent patients from unnecessary surgical intervention. I use the term advisedly, for it has been my experience that at no time has the patient benefited from

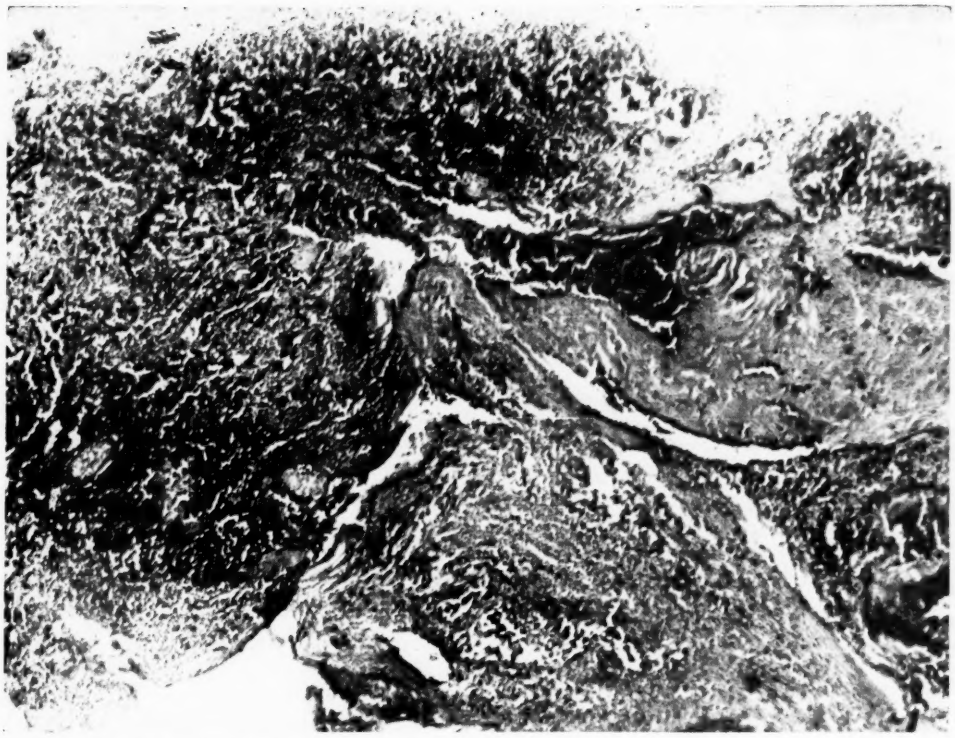
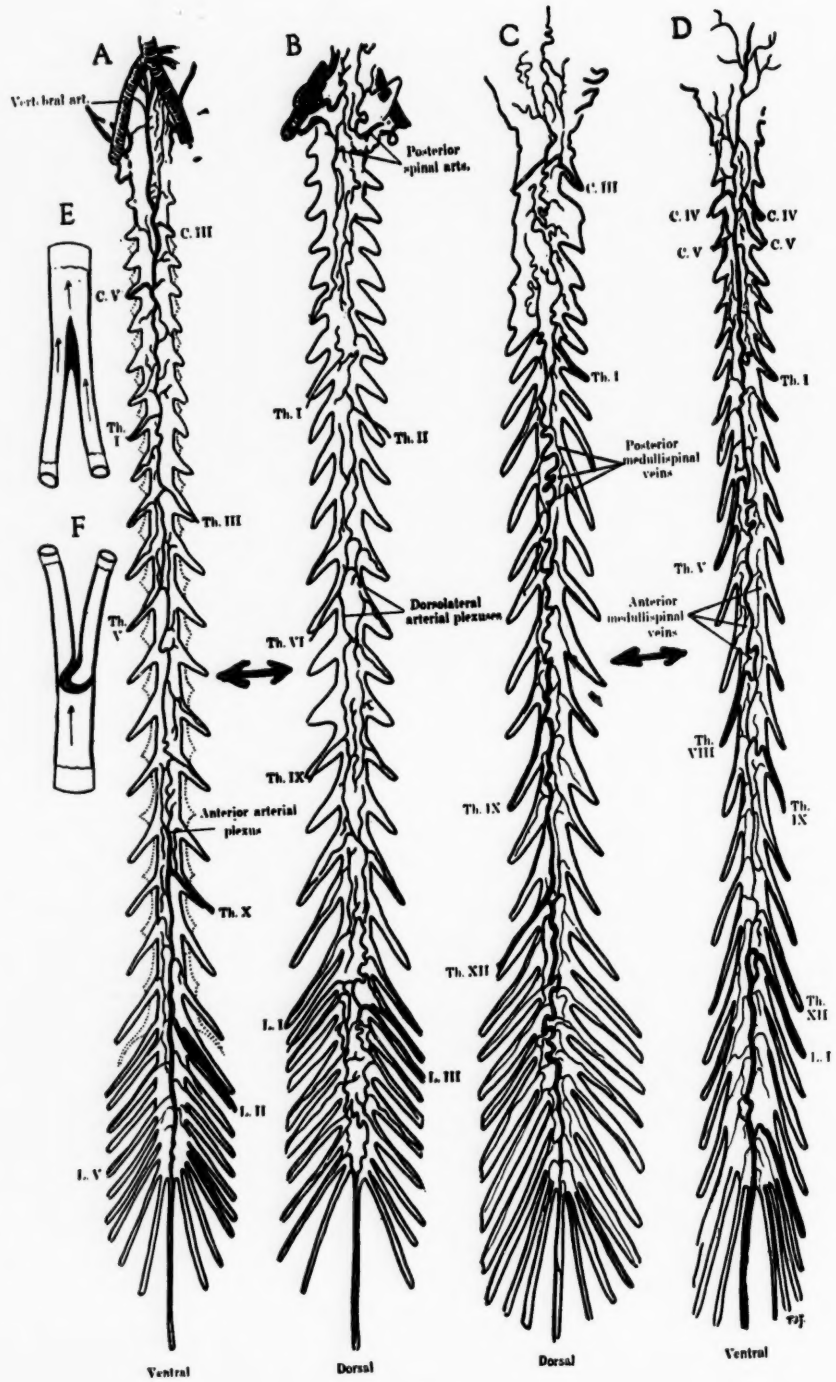


Fig. 1.—Spinal arachnoiditis from L. D. Note the dense collagenous tissue interspersed with areas of low grade inflammation. (Hematoxylin and eosin, × 1003.)

surgical exploration in the presence of chronic adhesive arachnoiditis. Indeed, I have been dismayed to discover, in reviewing my cases, that in those in which operation was performed the paraplegia was, for the most part, intensified.

How does chronic adhesive arachnoiditis produce its devasting effects upon neural structures within the spinal canal? One answer might be that it progressively chokes the axons traversing the area and the neuronal beds lying within it. This probably is not actually the case, for, as is well known, nerve tissue can stand a vast amount of compression over a long period without showing much disturbance of function. It is more likely that the damage done by arachnoiditis results from its effect upon the vascular supply to the spinal cord, attenuating the blood flow and starving the nervous tissues. The following case documents the validity of this concept of pathogenesis.

B. K., a 64-year-old man, was first seen in May 1949, complaining that for approximately one year he had noted progressive weakness in both legs. This had advanced to such a point that he could walk only with the greatest difficulty. Lately a troublesome constipation had appeared, and nocturia disturbed his sleep. For several years he had been treated for hypertension. When not under active treatment, his blood pressure in millimeters of mercury ranged in the neighborhood of 180 systolic and 90 diastolic. Under treatment with Priscoline®, however, the blood pressure was reduced to 110 systolic and 65 diastolic, and symptoms of paresis of the lower extremities became severe in a matter of hours. When therapy was relaxed and the blood pressure rose to 160 systolic and 80 diastolic, the weakness of the legs was promptly mitigated.


A
B
C
D
E
F
Vertebral art.
C. III
C. V
Th. I
Th. III
Th. V
Th. VI
Th. IX
Th. X
L. II
L. V
Anterior arterial plexus
Posterior spinal arts.
Th. II
Dorsolateral arterial plexuses
L. I
L. III
Th. XII
Posterior medullispinal veins
Anterior medullispinal veins
C. IV
Th. VIII
Ventral
Dorsal
Dorsal
Ventral

Treatment by a reducing diet and a rice diet produced the same results. Finally a compromise pressure of 145 systolic and 90 diastolic secured for the patient the optimum result.

The past history revealed that in February 1948, fifteen months earlier, he had noted a swelling in the right testicle. With the patient under spinal anesthesia, a hydrocele was discovered and corrected. It was some three months after this operation that the patient first began to have trouble with his legs.

On physical examination the blood pressure was 156 systolic and 90 diastolic and the cranial nerves were normal. Reflexes in the upper extremities were absent. The epigastric reflexes were present and active, while the lower abdominal and cremasteric reflexes were absent. Knee jerks and ankle jerks were present, the right patellar jerk being more brisk than the left. There was no response to plantar stimulation. There was approximately 50 per cent diminuation in tone, strength and speed of movement of the lower extremities, with slight atrophy, if any. Sensory examination revealed a definite sensory level at the ninth dorsal dermatome. This was present to testing with pin, ice, vibration and light touch. Joint sensibility was largely intact. Roentgenograms of the dorsal portion of the spine were completely normal. Spinal puncture revealed a total protein content of 100 mg. per hundred cubic centimeters, with the presence of three cells. Spinal manometric study did not reveal a block, but the fluid rose and fell slowly in the manometer on jugular compression. A myelogram was made, which showed an irregular stringing out and fragmentation of the iodized oil in the mid-dorsal region, past which oil flowed with difficulty on one side only.

On the basis of the history and the neurologic and myelographic observations, a diagnosis of spinal arachnoiditis was made. Surgical confirmation was not advised. The patient continued in a sedentary way of living until he had a stroke in 1951. This left him bedridden and with hemiplegia. At the time of writing he is in a nursing home.

Fig. 3.—Myelogram showing almost complete obstruction and characteristic streaking of iodized oil in the presence of spinal arachnoiditis.

This case provides strong inferential evidence of the constrictive effect of arachnoidal proliferations on the circulation of the spinal cord. Some degree of hypertension appears to have held in abeyance more serious damage due to arachnoiditis. This suggests that the pathologic physiologic condition illustrated here is the basis of the grave and usually irreversible neurologic consequences of chronic spinal arachnoiditis. Supporting evidence of this has been observed by Haymaker[16] in his studies on decompression sickness. He has noted that the brunt of the injuries borne by the nervous system in this disease fall upon the mid-dorsal region of the spinal cord and result from the relative paucity of nutrient vascular channels.

SUMMARY

Of the 9 patients observed by the author, 2 of whose cases are reported here

Fig. 2. (*opposite*).—Diagram of circulation of spinal cord, showing paucity of anastomotic channels in the neighborhood of T-6 (arrows). From F. A. Mettler, *Neuroanatomy, 2d ed.* 1948, courtesy of the C. V. Mosby Co., St. Louis, Mo.

in detail, all were between 40 and 65 years of age at the time of onset of their neurologic symptoms. Eight of the 9 were operated upon for pelvic or intra-abdominal disease and lay supine on the operating table or in a mild Trendelenburg position. The ninth, who had a hydrocele, though probably also flat, may have been tilted in the lithotomy position. The earliest neurologic symptoms appeared three months after the induction of spinal anesthesia; the latest, after two and one-half years.

All, as indicated before, had maximum neurologic levels in the neighborhood of the sixth dorsal, though frequently the greatest deficit was apparent at the lumbar and sacral levels. The clinical picture was that of a transverse myelitic lesion, as opposed to the "cauda equina syndrome" with lower motor neuron abnormalities. Four of the 9 patients with arachnoiditis were operated upon. Though no extensive resection was attempted, 3 of the 4 were worse, and 1 was certainly no better. Finally, all 9 patients recovered from their initial operations and anesthesia and left the hospital without neurologic symptoms. This is probably the most sinister aspect of this disorder, for those who performed the operations and gave the anesthetic are probably unaware that their services carried later reprisals.

CONCLUSIONS

1. Spinal anesthesia can cause late and vast neurologic damage by initiating a granulomatous, leptomeningeal proliferation.

2. This damage is unsuspected for some months after the induction of anesthesia.

3. It most frequently causes neurologic signs at the sixth dorsal level and suggests tumor.

4. The symptoms result from the constrictive effect of the arachnoiditis upon the vascular channels of the spinal cord, the least competent of which are in the mid-dorsal region.

5. Surgical intervention has small salvage value in such cases.

DISCUSSION BY DR. HAROLD C. VORIS, CHICAGO

It is a pleasure and a privilege to congratulate Dr. Williams on presenting this clear-cut anatomic and clinical syndrome of a serious late complication of spinal anesthesia. Certainly, from now on, when we are confronted with a patient with evidence of a lesion of the spinal cord at the sixth dorsal segment, we must think of a complication of spinal anesthesia and inquire into the past history of the patient to see whether, in fact, he has undergone spinal anesthetic. My own experience with complications of spinal anesthesia has been essentially with the first type of patient to which Dr. Williams referred, namely, the patient who shows immediate consequences due to the acute action of the anesthetic agent on the tissue of the spinal cord. In 1 such case exploration was done because the clinical picture was complicated by the fact that the patient had fallen from the cart to the floor during his transfer from the operating room to the ward. I think Dr. Williams will be glad to know that in that case at exploration, seventy-two hours after the initial operation and spinal anesthesia, marked opacity of the arachnoid was noted. There was no other gross abnormality. Biopsy of the arachnoid at that time showed intense round cell infiltration. This patient never showed any neurologic improvement, and he died eight weeks later. At necropsy the observations were comparable to those Dr. Williams described.

DR. WILLIAMS: I am very much interested in Dr. Voris' experience. He is one of the few who have had an opportunity to view the arachnoid so short a time after the administration of spinal anesthesia. Kansler has studied quite a number of cases of continuous spinal anesthesia and found cell counts running all the way up to 1,500 cells in an apparently otherwise uncomplicated case. Others have confirmed this work. She reports that in simple spinal anesthesia, when the needle is introduced and then withdrawn, the cell count goes up to maybe one or two hundred. It is my belief that it is the anesthetic agent itself that is responsible for this and not contamination of either the drug or the instruments.

QUESTIONS FROM THE FLOOR

DR. WATTS: Dr. Williams, do I understand from your presentation that you recommend the total abandonment of spinal anesthesia? Spinal anesthesia, as you know, is still very

popular in many of the hospitals in this country. If these are your recommendations and they are followed, it will mean the loss of a valuable tool in the service of the surgeon. Do you think that your observations justify this position?"

DR. WILLIAMS: Dr. Watts, spinal anesthesia is a useful agent. There are times when it is the only anesthetic agent available. I am thinking particularly of my experiences on shipboard during the last war. On more than one occasion I was called upon to administer an anesthetic in order to perform an operation and obviously couldn't do both at the same time. Spinal anesthesia used in such circumstances is unquestionably the anesthesia of choice. There are also times when no other form of anesthetic agent is available. Of course, it should then be used. As you know, surgeons are constantly weighing risks against gains. Today I point out that in a small but significant number of cases serious damage can result to the nervous system from the use of spinal anesthesia and my personal feeling is, speaking with profound interest in and respect for the nervous system, that spinal anesthesia should be reserved for those circumstances where other forms of anesthetic agents are for one reason or another inexpedient to employ.

DISCUSSION FROM THE FLOOR

DR. SAMUEL LOWIS: I should like to offer a few comments based on a series of 9 personally observed cases. These have not been studied as intensively as Dr. Williams' cases. One of these was in my care from its inception; the other 8 were patients whom I saw in consultation, and on 2 of whom I performed spinal exploratory operations.

In this series of 9 cases, the one common denominator was the fact that all of the patients had previously undergone spinal anesthesia before the one which led to the arachnoiditis. A closer questioning or study of 2 of these, including the one who had been in my care from the onset, showed that we might, with more careful attention to minor details in the history, have recognized an allergic or a toxic reaction to the earlier spinal anesthesia; this would have allowed us to avoid the later anesthesia which led to the serious complication.

A second difference between the observations in these cases and Dr. Williams' cases lies in the fact that in 3 of these the immediate appearance of neurologic signs was noted.

In 1 of these it was thought that the fault lay in contamination of an ampule. This patient never obtained anesthesia after the injection of Pontocaine; ether anesthesia had to be used for the operation, and paraplegia was present from the moment of administration of the drug.

The second patient had undergone a long series of operations for reconstruction of the common duct. In the second operation spinal anesthesia was used, apparently for the first time. There was slow recovery of strength in walking, and this was mistakenly attributed at the time to a prolonged shock reaction, thought to be due to inadvertent tear in the hepatic vein. At the last operation continuous spinal anesthesia was administered, with a level high enough for anesthesia of the diaphragm. I had occasion to explore the lumbar canal of this patient, and observed over an area of 3 cm. (the entire area of the exploration) a dense mass of scar tissue, binding all the nerves together and obliterating the subarachnoid space.

The third patient to be mentioned in detail presented a neurosurgical problem from the beginning. This patient had a ruptured disc, and later a recurrence and a need for refusion; both operations were done with Pontocaine anesthesia. While under anesthesia at the second operation the patient had some difficulty in respiration and a tingling in his arms, although the anesthesia did not ascend to this level, and noted also a prickling sensation in the tongue. For some moments he had to have assistance in respiration. This patient had had to be catheterized for five days after the first operation; little significance was attributed to this at the time, but in retrospect it seemed possible that it was related not to a mechanical difficulty in voiding but to a borderline difficulty related to the anesthesia.

Something may be said about the tissue changes on the basis of the single patient who was studied post mortem. This was the patient who had undergone many operations for disease of the gallbladder. A biopsy had been made at the operation; at autopsy, about three years later, there was observed not only scar tissue of the variety that might have been expected to compress the blood vessels, as Dr. Williams suggested, but infiltration of the scar tissue into the cord to a depth of several millimeters. It seemed to me that in some cases, at least, this may explain the pathogenesis of this lesion. Certainly in most cases the anesthetic may well pool at the sixth dorsal

level, but the patient who had the two operations for disc disease was never in the supine position, and if pooling of the anesthetic was a factor the greatest concentration must have been at the first and second lumbar level.

The problem as to whether this is a toxic or an allergic reaction needs further study. It seems to me, however, that with a relatively higher incidence of serious complications following secondary or later spinal anesthesia, one should whenever possible avoid exposing the patient to the additional risks associated with such occasions.

CLOSING REMARKS BY DR. WILLIAMS

DR. WILLIAMS: Dr. Lowis described particularly 3 cases which I purposely eliminated from my presentation. These are cases in which the immediate effects of the anesthesia upon the nervous system were apparent. This is the "cauda equina syndrome," which is reported in the vast majority of papers in the literature today in referring to neurologic consequences of spinal anesthesia. It is the acute toxic or allergic, if you please, reaction occurring within minutes or hours after the administration of the anesthetic and even producing 6th nerve palsies and other cranial nerve palsies such as tingling in the tongue as happened in one of Dr. Lowis' cases. Pooling is apparently not important in untoward developments such as these, and consequently this explanation need not be involked to account for the persistence of neurologic abnormalities in that patient of Dr. Lowis' who had a laminectomy and was operated upon in the prone, not the supine, position. With regard to the question whether subtle neurologic abnormalities are really present in those chronic cases though undetected at the time of discharge, it is highly difficult to say. Customarily a neurologic examination is not done upon every patient who has had, for example, a gallbladder taken out, and neither the general surgeon or the anesthesiologist at the time of the discharge is alarmed by any complaints that the patient may make. The patient is apparently well and as far as he and everybody else is concerned, free of trouble.

RÉSUMÉ

Les neufs patients observés par l'auteur, dont deux cas sont rapportés en détail, étaient âgés entre 40 et 65 ans au moment de l'apparition des symptômes neurologiques. Huit de ces 9 cas avaient été opérés pour une affection pelvienne ou intra-abdominale, couchés sur le dos ou en léger Trendelenburg. Le neuvième malade, atteint d'un hydrocèle, avait été probablement opéré en position couchée ou en position genu pectorale. Les premiers symptômes neurologiques apparurent trois mois après l'anesthésie rachidienne; les derniers furent signalés deux ans et demi après.

Comme l'auteur l'a décrit, le maximum de symptômes neurologiques siégeaient au niveau de la sixième vertèbre dorsale, quoique fréquemment les symptômes les plus graves se localisèrent au niveau de la région lombaire et sacrée. L'image clinique était celle d'une lésion myélitique transversale s'opposant à un syndrome de la queue de cheval. Quatre des neuf malades avec arachnoïdite furent opérés. Bien qu'aucune résection extensive ne fut pratiquée, l'etat de trois des quatre malades empira et le quatrième resta stationnaire. Finalement les neuf malades guérirent de leur première opération et des complications survenues à la suite de l'anesthésie. Ils quittèrent l'hôpital sans symptômes neurologiques. Ces sortes de complications post-opératoires sont, pour le chirurgien, des plus décevantes.

CONCLUSIONS

1. L'anesthésie rachidienne peut causer des troubles neurologiques tardifs et importants en produisant une prolifération granulomateuse, au niveau des méninges.

2. Aucun symptôme ne se manifeste durant quelques mois après l'anesthésie.

3. Les signes neurologiques surviennent le plus fréquemment au niveau de la sixième vertèbre dorsale et font penser à une tumeur.

4. Les symptômes proviennent des effets spastiques des vaisseaux situés au niveau de la moelle (arachnoïdite), dont les moins développés siègent au niveau de la région dorsale.

5. Dans ces cas, l'intervention chirurgicale n'a pas d'effet.

ZUSAMMENFASSUNG

Alle neun vom Verfasser beobachteten Kranken, von denen er zwei hier ausführlich schildert, befanden sich zur Zeit des Auftretens der neurologischen Symptome in Alter zwischen 40 und 65 Jahren. Von den neun Kranken wurden acht wegen Becken-oder Baucherkrankungen operiert und lagen auf dem Operationstisch in flacher Rückenlage oder in leichter Trendelenburgscher Lage. Der neunte hatte eine Hydrozele und lag wahrscheinlich auch flach, kann aber vielleicht in Blasenschnittstellung gedreht worden sein. Die ersten neurologischen Symptome traten drei Monate nach Enleitung der Rückenmarksanästhesie, die spätesten nach zweieinhalb Jahren auf.

Bei allen Patienten gehörten die maximalen neurologischen Symptome der Nachbarschaft des sechsten Dorsalsegments an, wenn auch häufig die grössten Ausfälle in der Höhe des Lenden-und Sakralmarks in Erscheinung traten. Das klinische Bild war das einer Querschnittsmyelitis im Gegensatz zum "Cauda equina-Syndrom" mit Anomalien des unteren motorischen Neurons. Vier der neun Kranken mit Arachnoiditis wurden operiert. Obgleich kein Versuch einer umfangreichen Resektion unternommen wurde, erfuhren drei von den vier eine Verschlechterung, und der vierte sicherlich keine Besserung. Alle neun Patienten überstanden die ursprüngliche Operation und Anästhesie gut und verliessen das Krankenhaus ohne neurologische Erscheinungen; und darin liegt wohl das Tückischste dieser Erkrankung, dass diejenigen, die die Operation ausführten und die Anästhesie verabfolgten, wahrscheinlich garnicht wissen, zu welchen Nackenschlägen ihre Handlungen später führten.

SCHLUSSFOLGERUNGEN

1. Die Spinalanästhesie kann zu granulomatösen leptomeningealen Wucherungen führen und auf diese Weise späte und umfangreiche neurologische Schädigungen hervorrufen.

2. Mehrere Monate lang nach Einleitung der Anästhesie bestehen keine Verdachtsmomente für die Schädigung.

3. Meistens kommt es zu neurologischen Krankheitserscheinungen in Höhe des sechsten Dorsalsegments, die den Verdacht auf Tumor erwecken.

4. Die Symptome sind die Folge einer Einschnürung der Gefässe des Rückenmarks durch Arachnoiditis; die schwächsten Gefässe liegen in der Gegend der mittleren Brustwirbelsäule.

5. Chirurgische Behandlung ist in diesen Fällen von geringem Wert.

RESUMEN

De 9 pacientes observados por el autor, 2 de ellos comunicados en detalle, todos estuvieron entre los 40 y 65 años de edad al comienzo de sus síntomas neurológicos. Ocho de los 9 fueron operados de padecimientos pélvicos ó abdominales y permanecieron en posición supina sobre la mesa de operaciones ó bien en moderado Trendelenburg. El noveno que tenía un hidrocele aun cuando probablemente plano pudo haber sido inclinado en posición de litotomía. Los primeros síntomas neurológicos aparecieron tres meses después de una inducción con anestesia raquídea; el último después de dos años y medio.

Como se indicó anteriormente todos tuvieron niveles neurológicos máximos en la vecindad de la sexta dorsal, aun cuando el déficit mayor fué en niveles lumbares y sacros. La imagen clínica fué la de una mielitis transversa, antagónicamente al síndrome de "cauda equina" con anormalidades de motoneurona inferior. Cuatro de los 9 pacientes con aracnoiditis fueron operados. Aun cuando se intentó una resección masiva, 3 de los 4 empeoraron y 1 lo hizo en una forma definida. Finalmente, los 9 pacientes se recuperaron de la operación inicial y anestesia y dejaron el hospital sin síntomas neurológicos. Esto es uno de los aspectos más siniestros de esta perturbación ya que aquellos que administran los anestésicos y operan no se dan cuenta de que sus servicios acarrean represalias.

CONCLUSIONES

1. La anestesia raquídea puede ser una causa de amplias lesiones neurológicas tardías produciendo proliferación granulomatosa leptomeníngea.

2. Este lesión pasa desapercibida por algunos meses después de la inducción anestésica.

3. La causa más frecuente de signos neurológicos se encuentra a nivel de la sexta dorsal y sugiere tumor.

4. Los síntomas resultan del efecto constrictivo de la aracnoiditis sobre los conductos vasculares de la médula espinal, que son menos competentes en la región media torácica.

5. La intervención quirúrgica tiene un valor muy limitado en tales casos.

RIASSUNTO

Dei 9 pazienti studiati dal l'autore, 2 dei quali riferiti dettagliatamente, tutti avevano un'età fra i 40 e i 65 anni al momento dell'insorgenza della sintomatologia neurologica. Su 8, dei 9, era in corso un intervento per stati patologici intraaddominali o pelvici e giacevano sul tavolo operatorio supini o in posizione di Trendelemburg. Il nono, che aveva un idrocele, doveva essere in posizione di litotomia. I primi sintomi neurologici apparvero a tre mesi di distanza dall'esecuzione dell'anestesia spinale; i tardivi dopo 2 anni e mezzo. Come si è già detto, la massima altezza del livello neurologico era in tutti i casi sulla sesta dorsale, per quanto il deficit più grave era frequentemente ai livelli lombare e sacrale. Le manifestazioni cliniche erano quelle di una mielite trasversa, in opposizione alla "sindrome della cauda equina" con disturbi del neurone motorio inferiore. Quattro dei nove pazienti con aracnoidite furono operati; ma, per quanto non fosse stata eseguita una dissezione estesa, tre dei quattro peggiorarono e uno non migliorò certamente. Alla fine, però, tutti l 9 pazienti lasciarono l'ospedale senza disturbi neurologici. L'aspetto forse peggiore di tali sindromi post-anestetiche è rappresentato dal fatto che i chirurghi e gli anestesisti che originariamente eseguirono gli interventi e le anestesie non furono probabilmente informati che la loro opera era stata tardivamente seguita da disturbi neurologici.

CONCLUSIONI

1. La rachianestesia può causare lesioni neurologiche gravi e tardive, dando origine a processi granulomatosi leptomeningei.

2. Tali lesioni passano inosservate per alcuni mesi dopo l'esecuzione della rachianestesia.

3. Esse, il più di frequente, producono disturbi neurologici a livello della sesta dorsale e fanno pensare ad un tumore.

4. I sintomi sono caucati dallo strozzamento da parte dell'aracnoidite, della rete vascolare del midollo spinale—che è appunto funzionalmente meno valida nella regione dorsale media.

REFERENCES

1. Davis, L.; Haven, H.; Given, J. H., and Emmet, J.: Effects of Spinal Anesthetics on the Spinal Cord and its Membranes, J.A.M.A. **97**: 1781, 1931.

2. Lundy, J. S.; Essex, H. E., and Kernohan, J. W.: Experiments with Anesthetics: IV. Lesions Produced in the Spinal Cord of Dogs by a Dose of Procaine Hydrochloride Sufficient to Cause Permanent and Fatal Paralysis, J.A.M.A. **101**: 1546, 1933.

3. Brock, S.; Bell, A., and Davison, C.: Nervous Complications Following Spinal Anesthesia, J.A.M.A. **106**:441, 1936.

4. Ferguson, F. R., and Watkins, K. H.: Paralysis of the Bladder and Associated Neurological Sequelae of Spinal Anesthesia (Cauda Equina Syndrome), Brit. J. Surg. **25**:735, 1937.

5. Critchley, Macdonald and others. Discussion of the Neurological Sequelae of Spinal Anesthesia, Proc. R. Soc. M. London **30**:1007, 1937.

6. Kennedy, F.; Effron, A., and Perry, G.: The Grave Spinal Cord Paralyses Caused by Spinal Anesthesia, Surg., Gynec. & Obst. **91**:385-398, 1950.

7. Kennedy, F.; Somberg, H. M., and Goldberg, B. B.: Arachnoiditis and Paralysis Following Spinal Anesthesia, J.A.M.A. **129**:664, 1945.

8. Editorial: The Method was Good but the Patient Died, Anesthesiology **11**:254-255, 1950. Cole, F.: A Defense of Spinal Anesthesia, Anesthesiology **13**:407-415, 1952.

9. Winkleman, N. W.: Neurologic Symptoms Following Accidental Intraspinal Detergent Injection, Neurology **2**:284-291, 1952.

10. Lewis, L. D.: Storing Ampules for Spinal Anesthesia, J.A.M.A. **153**:50, 1953.

11. Searles, P. W., and Nowill, W. K.: The Role of a Sterilizing Solution in the Cauda Equina Syndrome Following Spinal Anesthesia, New York State J. Med. **50**:2541, 1950.

12. Kamman, G. R., and Baker, A. B.: Damage to the Spinal Cord and Meninges Following Spinal Anesthesia, Minnesota Med. **26**:786, 1943.

13. Lindemulder, F. G.: Spinal Anesthesia; Its Effect on the Central Nervous System, J.A.M.A. **99**:210, 1932. Rabiner, A. M.: Concerning Neurologic Complications Following Spinal Anesthesia, New York State J. Med. **50**:2546, 1950.

14. Haynes, W. G., and Smith, F. A.: Cervical Arachnoiditis Occurring After Spinal Anesthesia, Anesthesiology **13**:444, 1942.

15. Kamsler, P. M.: Study of Changes in Spinal Fluid Cell Count During Spinal Anesthesia. Cur. Researches in Anes. and Anat. **30**:103, 1951

16. Haymaker, W.: Personal communication.

A very unusual use for which the microscope was employed is reported from England. In Yorkshire there is an ancient church which dates from the earliest period of English history. On the rough oak door of this church were some fragments of material, hardened and blackened by time, nailed there with heavy spikes. According to a tradition almost as old as the church, these bits of hardened material were the remnants of the skin of an early Danish invader who had attempted to violate the sanctity of the church, but had been captured by the irate villagers, skinned alive, and his skin nailed to the church door as a warning to trespassing, blaspheming Danish pirates.

A certain curious person, to test the truth of this legend, took a piece of this material and examined it under a microscope. This examination showed that when the formless piece of material was magnified under the microscope, cells were very clearly seen. Further study showed that it was really a piece of skin, and that on its surface were small blond hairs, characteristic of the early invaders of England. Thus the venerable tradition was substantiated to the degree at least of proving that it was human skin, belonging to a person with light hair, and very probably a Dane.

—*Major*

Spinal Cord Reflexes

KARL-ERIK HAGBARTH, M.D., D.A.B.

STOCKHOLM, SWEDEN

IT IS not the purpose of this presentation to describe several kinds of spinal reflexes. Instead, I prefer to discuss the problem of how spinal reflexes in general can be modified and regulated from higher levels of the nervous system.

First, it may be necessary to define spinal cord reflexes. Of course, they are reflexes mediated by the spinal cord itself, or, in other words, the reflex arcs are restricted to the spinal cord and do not extend into higher levels. But how does one find out whether a reflex belongs to this group or not? Sometimes the latency of the reflex is short enough to tell the story. But if the latency is longer the method is to find out whether the reflex can be mediated by a spinal cord completely separated from higher levels of the nervous system.

It must be remembered, however, that an isolated spinal cord is working under very unphysiologic conditions, and in the normal intact animal the spinal cord reflex may be impossible to evoke, because of tonic descending influences from above which normally maintain a depression of the spinal neurons involved. Similarly, there may be spinal reflexes that cannot be mediated by an isolated spinal cord because the spinal neurons involved in the reflex arc depend for their normal function upon tonic facilitatory influences from higher centers.

Thus, it seems evident that, in order to obtain a better understanding of spinal reflexes, one must learn more about the descending pathways from above that normally influence the activity in the spinal cord.

Read at the Eighteenth Annual Congress of the United States and Canadian Sections, International College of Surgeons, New York, Sept. 13-17, 1953.
Submitted for publication April 24, 1954.

Let us start with the relatively simple monosynaptic stretch reflex and see how this reflex can be influenced from higher centers. The sense organs from which the reflex originates are the muscle spindles. When a muscle is stretched—as, for instance, when one elicits the knee jerk—the stretch applied upon the muscle spindles brings about an afferent discharge from these endorgans. This sensory discharge is conducted in the afferent fibers connecting the muscle spindle with the spinal cord. Many of these spindle afferents pass directly through the spinal cord and reach the motor neuron in the ventral horn without involving any intermediate neurons in the spinal cord. Thus the only synaptic junction in this reflex arc is the one on the motor neuron. If the afferent influx from the muscle spindle is strong enough the motor neuron is brought into activity and motor impulses are conducted in the efferent nerves to the muscle that contracts.

This monosynaptic stretch reflex, which forms the basis of other postural reflexes, can be interfered with and regulated in many different ways. Thus, it is known that pyramidal fibers can influence the excitability of the motor neurons. These descending fibers, however, do not make direct contact with the motor neurons. They are connected to interneurons in the spinal cord, and thus they reach the motor neurons in an indirect way. Destruction of the pyramidal fibers causes paralysis but no spasticity, no exaggeration of the stretch reflex and no increased tonus in the muscles. There is no doubt that corticospinal fibers are mainly concerned with the regulation of voluntary movements. Tremor and other involuntary movements in man, however, are also abolished after section of the pyramidal tract. This does

not necessarily mean that the repeated, synchronized, descending volleys responsible for tremor arise in the motor cortex and are conducted to the motor neurons by the pyramidal tract. It seems more likely that the corticospinal tract contributes such an essential facilitative background for motor neuron activity that the pathologic, descending synchronized volleys from other sources are unable to reach the threshold in its absence. This question, however, is an important one, for its answer should determine whether or not it is possible to relieve involuntary movements by neurosurgical methods in man without, at the same time, paralyzing the patient.

The excitability of the motor neuron is influenced not only by the corticospinal fibers but by the descending extrapyramidal pathways. During recent years special attention has been drawn to the reticulospinal connections that obviously emerge as the major final common paths of the extrapyramidal motor system. Not only the cerebral cortex and the cerebellum but the basal ganglia utilize reticular relays in the brain stem in exerting many of their influences upon the cord. Like the pyramidal fibers, most of the reticulospinal fibers do not make synaptic connection directly with the motor neuron, but spinal interneurons are involved in the transmission.

The effect of the reticular formation upon the spinal motor neurons depends upon what region of the reticular formation that is activated. Stimulation in the lower bulbar part of the reticular formation usually inhibits or diminishes the excitability of the motor neurons, but stimulation in the midbrain reticular formation usually causes an increase in their excitability.

The inhibitory bulbar reticular formation receives connections from the suppressor areas of the cerebral hemispheres and the cerebellum. Injury to the suppressor areas in the cat produces spasticity, evidently because of the fact that the inhibitory bulbar reticular formation is left without inflows to maintain its activity. The typical feature of this spasticity is exaggeration of the stretch reflexes in all muscle groups of the extremity, but most pronounced in the functional extensors. The exaggeration of the monosynaptic stretch reflex, however, is not due to an intrinsic ability of the motoneurons to increase their excitability when released from suppression. Their increased excitability is due to facilitative influx from the brain, normally balanced by suppression, but exerting an augmented influence in its absence. Thus a preexisting spasticity can be greatly diminished by lesions in the facilitative midbrain reticular formation. The results indicate, however, that the reticulospinal facilitatory discharge is the major but not the sole influx maintaining spasticity. Some is derived also from the vestibular nuclei, injury to which, when added to midbrain reticular destruction, eliminates residual spasticity.

These observations clarify the results of neurosurgical attempts to relieve spasticity in man by section of facilitatory paths in the cord. Spasticity is not prevented by section of vestibulospinal tracts in the human cord. The facilitatory reticulospinal pathways must also be transected. These facilitatory paths maintaining spasticity are concentrated in the ventral half of the cord, but their widespread distribution suggests that effective interruption might leave the patient with spasticity relieved but more incapacitated than before.

Hypokinesia, as well as involuntary movements, can be produced experimentally in animals after the occurrence of lesions in the midbrain reticular formation or in extrapyramidal structures connecting with it. Much of the immobility of hypokinesia, however, appears due to lack of will to move rather than to paralysis of movement. Thus, while reduction of excitation within the cord may be in part responsible for the effect, there are indications that a defect at the cerebral level may also be involved, showing that the affected part of the brain stem exerts influence upon the upper as well as the lower levels of the nervous system.

So far we have been interested only in

suprasegmental influences upon the motor neuron, that is, the efferent part of the monosynaptic stretch reflex. Quite recently it has been shown, however, that the afferent link in this reflex is also under the influence of descending pathways from higher levels. It has long been known that among the efferent nerve fibers in the ventral root which innervate the motor units in the muscles there are smaller efferent fibers which also pass down to the muscles. Now it has been definitely proved that in mammals the only function of these small, so called gamma efferent nerve fibers is to innervate the muscle spindles. Thus activity in the gamma fibers does not cause any contraction of the muscle but an increased afferent discharge from the muscle spindles. It has also been demonstrated that the efferent gamma system is tonically activated from central regions and that stimulation of various central nervous structures influences the afferent discharge from the muscle spindles. One main difference between the activity of the gamma fibers and the ordinary motor neuron discharge is this: In normal rest, when the muscles are completely relaxed, the ordinary motor neurons are silent; there is no spontaneous activity going on in the motor units; but there is constant background activity in the small gamma efferents, which thus maintains a certain degree of excitability in the muscle spindles. Since the afferent discharge of the muscle spindles has a marked effect on the excitability of the ordinary motor neurons, it is obvious that one is here dealing with an external circuit by means of which higher levels of the nervous system, in an indirect way, can regulate the excitability of the motor neurons.

The most powerful inhibitory effect on the activity of gamma efferents and spindle afferents was obtained by stimulating the bulbar reticular formation, and the most powerful excitatory effect is obtained from the midbrain reticular formation. Thus it is obvious that extrapyramidal pathways are those mainly responsible for the suprasegmental regulation of the efferent gamma system, but it is not unlikely that pyramidal fibers also are capable of influencing the gamma efferent discharge. The clinical significance of the whole gamma system is still unknown, but there is no doubt that it plays an important role in the regulation of tonic muscular activity. It is also worth pointing out that transection of a dorsal root can no longer be regarded as a strictly peripheral affair. Actually, this operation deprives the spinal cord of some of the descending influences from higher levels.

This has been a short description of how the monosynaptic stretch reflex can be influenced from higher levels. It still remains to describe how other spinal cord reflexes which are not monosynaptic but polysynaptic can be regulated from above. Of course, a polysynaptic reflex is one in which the reflex arc includes a series of interneurons between the sensory and the motor side, and a typical spinal polysynaptic reflex is, for instance, the flexor reflex. Since the motor neuron in the ventral horn is the only final common pathway for all spinal reflexes, it is evident that descending pathways capable of influencing the excitability of these ventral horn cells have an effect also upon all spinal reflexes, monosynaptic as well as polysynaptic. But the problem is whether a polysynaptic reflex can be interfered with not only at the motor neuron junction but also at some early synapses in the reflex arc. The answer to this question is important, not only for a better understanding of the suprasegmental regulation of spinal reflexes but for understanding of the mechanisms involved in transmission of afferent impulses within the cord.

It is known that many ascending afferent pathways in the spinal cord are made up by branches of the same interneurons that participate in spinal polysynaptic reflexes. Thus, if one supposes that the synaptic junction between the incoming afferent fiber and the first interneuron in the spinal cord is under the physiologic influence of descending pathways, one has the necessary premises for suprasegmental regulation not only of spinal polysynaptic reflexes but of afferent

conduction in the spinal cord. This problem has recently been investigated by neurophysiologic methods in cats, and the results indicate that sensory relays in the spinal cord are normally under a tonic influence from higher levels and that stimulation of extrapyramidal structures in the brain can inhibit the afferent synaptic transmission in the spinal cord. As yet it is not known whether the pyramidal tract also has a regulating effect upon the sensory side of the spinal cord, but it does not seem unlikely.

Much work remains to be done before the intimate connections between the spinal cord and the rest of the nervous system are fully understood. There is no doubt, however, that the more one learns about these connections the better one will be able to interpret the mechanism behind many clinical manifestations of disturbance in the central nervous system.

Discussion by Dr. Grayson P. McCouch, Philadelphia

Dr. McCouch: I am glad that, in Dr. Hagbarth's lucid presentation of recent work on the influence of descending pathways, he has stressed the fact that there are many problems still unsolved.

Of these, one of the more obvious is the influence of the reticular formation upon decerebrate rigidity. A transection extending from the intercollicular region dorsally to the superior margin of the pons ventrally removes the major portion of Magoun's falicitatory area, leaving most of his inhibitory area intact. Yet the result is a release of excess extensor activity with inhibition of flexor neurons. The same result may be produced by relatively small bilateral lesions just internal to the red nuclei. As Dr. Hagbarth has stated, the rigidity is maintained in part through the vestibulospinal tracts. Yet it may persist after either acute or chronic destruction of both labyrinths. An answer is suggested by results recently reported by Sprague and Chambers, who have noted that weak, sharply localized stimuli in either the facilitatory or the inhibitory area give reciprocal effects.

Dr. Hagbarth has stressed the quantitative changes resulting from removal of descending impulses. There are also qualitative changes. Of these the most familiar is in the plantar reflex. When the large corticospinal supply to the foot is cut off, the spinal centers employ the long muscles to the digits (muscles that span two joints) for the gross purpose of ankle flexion, without regard to their effect upon the toes. With a sufficiently painful stimulus, dorsiflexion of the toes may be elicited even in the normal subject, the primitive spinal type of response dominating cerebral control.

One of the most fascinating observations Dr. Hagbarth has reported is the evidence that sensation may be inhibited at the spinal level by descending impulses. With characteristic modesty, Dr. Hagbarth refrained from stating that this is his own most recent contribution.

Questions from the Floor

Question: Is there any evidence that the efferent gamma system is present in man, and, if so, is it known that it has the same function as in the cat?

Dr. Hagbarth: It is known that efferent gamma fibers exist also in man. I don't think any clinical experiments have been done to elucidate the function of these fibers, but it seems quite reasonable to assume that the fibers, have a similar function in cat and in man.

Question: Are the efferent gamma fibers in the ventral root in any way separated from the ordinary motor fibers? Would it not be possible, for instance, to transect the gamma fibers without influencing the innervation of the ordinary muscle fibers?

Dr. Hagbarth: In the ventral root the gamma fibers are all intermingled with the ordinary motor fibers. So far there is no method for selective destruction of either of these two fiber systems.

Zusammenfassung

Der Verfasser erörtert die Modifizierung und Regulierung spinaler Reflexe von höher gelegenen Zentren des Nervensystems aus. Die Rückenmarksreflexe werden definiert als solche, die nach völliger Abtrennung von höher gelegenen nervösen Zentren vom Rückenmark vermittelt werden können; es wird allerdings darauf hingewiesen, dass Hemmung durch tonis-

che Einflüsse oder Ausbleiben erleichternder Einflüsse von oben die Auslösung des Rückenmarksreflexes unmöglich machen können. Der Verfasser erklärt die Art und Weise, in welcher der Reflex von höheren Zentren reguliert wird, und benützt den verhältnismässig einfachen monosynaptischen Streckreflex als Beispiel. Auf die Erörterung polysynaptischer Reflexe, z. B. des Beugungsreflexes, wird nicht eingegangen; allerdings bemerkt der Verfasser, dass absteigende Nervenbahnen, die imstande sind, die Erregbarkeit der Vorderhornzellen zu beeinflussen, einen Effekt auf alle Rückenmarksreflexe ausüben. Das Problem ist, ob ein polysynaptischer Reflex nicht nur an der Schaltstelle des Motorneurons sondern ausserdem noch an einer früheren Synapsis im Reflexbogen unterbrochen werden kann. Die Beantwortung dieser Frage ist von Bedeutung nicht allein zum besseren Verständnis der Regulation von Rückenmarksreflexen sondern auch zur Erklärung des Mechanismus, der bei der Übertragung afferenter Impulse innerhalb des Rückenmarks eine Rolle spielt.

RESUMEN

Los autores discuten la modificación y regulación de los reflejos medulares de los niveles superiores del sistema nervioso. Los reflejos medulares se definen como aquellos que pueden ser mediados a traves de la médula espinal completamente separada de los niveles superiores, aunque se señala que por influencia tónica ó ausencia de acción facilitarora superior puede hacerse imposible la producción de los reflejos medulares. Usando al reflejo monosináptico por estiramiento como ejemplo, el autor explica la manera por la cual es regulado por los centros superiores. No se entra en la discusión de los reflejos polisinápticos tales como los reflejos flexores; sin embargo el autor señala que las vías descendentes influenciando la excitabilidad de las células de las astas anteriores pueden tener efecto sobre todos los reflejos espinales. El problema es de si puede interferirse un reflejo polisináptico en una sinapsis anterior a la motoneurona en el arco reflejo. La contestación a esta pregunta es importante no solamente para el mejor conocimiento de la regulación de los reflejos espinales sino para el entendimiento de los mecanismos envueltos en la trasmisión de los impulsos medulares aferentes.

RIASSUNTO

L'autore tratta della modificazioni e della regolazione dei riflessi spinali da parte delle sezioni più alte del sistema nervoso. Per riflessi midollari si intendono quelli che si compiono in un midollo completamente separato dal resto del sitema nervoso, pur tenendo presente che tali riflessi posoono essere aboliti per la sovrapposizione di influssi tonici o per la mancanza di influssi favorenti da parte delle sezioni superiori. Servendosi come esempio del riflesso monosinaptico, che è abbastanza semplice, l'Autore spiega in qual modo esso sia regolato dai centri superiori. Non entra invece nella spiegazione dei riflessi polisinaptici, i flessori ad esempio, benchè le vie discendenti, capaci di influenzare l'eccitabilità delle cellule delle corna anteriori, possano avere effetto su tutti i riflessi spinali. Il problema verte sulla possibilità che un riflesso polisinaptico possa essere influenzato non soltanto alla giunzione del neurone motore ma anche in qualche altra sinapsi dell'arco riflesso. La risposta a ciò è importante non solo per una miglior comprensione della regolazione dei diflessi spinali, ma anche per comprendere i meccanismi in giuoco della trasmissione degli impulsi afferenti nell'interno del midollo.

Spinal Echinococcosis

FEYYAZ BERKAY, M.D., F.I.C.S.*

ISTANBUL, TURKEY

ECHINOCOCCOSIS involving the central nervous system is rarely seen in European and North American countries. This condition is not infrequent in South America, Australia and Iceland. During my four-year period in St. Louis, in visiting many neurosurgical clinics, I never encountered a case of cerebromedullary echinococcus, nor has Dr. Roland M. Klemme of St. Louis, my former chief, mentioned one cerebral echinococcus in his whole collection. Dr. German of New Haven, Connecticut, stated one year ago that he and his associates had had 1 case, but it was not definitely proved. In 1952 Dr. Kean of the Department of Tropical Medicine of Cornell, while visiting Istanbul, was present when I removed a calcified hydatid cyst of the brain. He stated at that time that this type of lesion is very rare in the United States and suggested that I bring my personal collection to a future Congress. My former chief, Dr. Klemme, encouraged this suggestion.

This paper represents a series of cases of cerebrospinal echinococcosis that have come under my care in the past two and one-half years in Istanbul. Six cases, in 4 of which the brain was affected, and in the other 2 the spinal cord and spine, are here reported. This is an unusual number even in my country, and I am delighted to say that all the patients were surgically treated and the condition entirely cleared up.

According to recent statistics given by F. A. Yücell (Hastene N. 1, 1953) in a series of 3,670 autopsies performed in Bakirkoy Mental Hospital, only 4 cases of intracranial echinococcosis had been observed, and all of the deaths had resulted from this disease of the brain. In addition, the Department of Pathology of Istanbul University, in over 10,000 necropsies, did not encounter more than 3 cases of echinococcosis of the nervous system. At the time of writing I have not encountered any published case of spinal echinococcosis in the Turkish medical literature.

During the past fifteen years, in the First Surgical Clinic of Istanbul University, it has become evident that Turkey is one of the countries where echinococcosis is a common parasitic disease. Practically all of the patients have been seen by me, particularly those in whom the infestation was cerebral and spinal. The majority of patients with this condition usually have involvement of the liver and lungs and, less frequently, of other organs. In this series the central nervous system is the primary site, and no involvement of other organs was observed.

The echinococcus infesting the central nervous system is the larva of the Taenia echinococcus of dogs, cats and sheep. Ingested eggs of this taenia represent the embryonic stage of the parasite, which penetrates and perforates the intestinal wall. From there, via the blood stream and the lymphatics, it spreads to the different organs, usually by way of the portal stations in the liver, and develops the well known hydatid cyst. To reach other organs of the body the embryo of the parasite passes through the capillaries of the liver and lungs, which accounts for the rarity of the condition in other parts of the body. Dévé's statistics show that in no more than 1 per cent of cases were other organs involved, and Lombard's statistics show the incidence to be less than 3 per cent. In the greatest number of cases reported the condition has originated in Iceland, Australia and South Africa (Cob-

*Assistant Professor of Clinical and Neurological Surgery, Istanbul University Faculty of Medicine.

Submitted for publication April 24, 1954.

bod). Henneberg reported the number of European cases of echinococcus infestation as less than 120. Morquio has collected 38 cases in twenty-five years. My series, collected in less than two and one-half years would suggest that the yearly incidence is twice that of Morquio's experience.

Echinococcosis of the Spine and Spinal Cord (Spinal Hydatidosis).—Two cases of spinal echinococcosis are here reported in which the patients were treated surgically by myself in the past two and one-half years. The Turkish medical literature at present would suggest that this condition has not appeared in the past. Consequently, spinal echinococcosis is a rarity even in Turkey. Purely intraspinal and intramedullary localization of the echinococcus is exceptional. In most cases one observes extramedullary, intradural or extradural, spinal or paraspinal localization of the hydatid cysts. Paravertebral soft tissues, muscles and vertebrae are involved and invaded by parasitic cysts and echinococcus lesions. In all cases of spinal echinococcosis in this series the spinal cord and meninges were involved and compressed directly by the cysts.

This spinal phenomenon appears in various forms. The hydatid cyst contains a clear fluid and numerous vesicles. Ordinarily the echinococcus is a multivesicular or mastic aseptic liquid surrounding the cyst. In Case 1, in which the lesion was removed from the lower lumbar region, the gross appearance of the surgical specimen simulated ground beef (Fig. 4). In Case 2, with the lesion in the upper cervical region, the appearance was that of systemic hydatidosis (Fig. 7). There were several cysts of different sizes and very little intercystic gelatinous material.

Spinal echinococcosis in the thoracic portion of the spine may involve the mediastinum, the pleura or the ribs as well as the paravertebral structures. Cervical echinococcuosis involving the surrounding structures may simulate cystic tumor or, as in my second case, may strongly suggest malignant change. Careful differential diagnosis and study of the lesion are important. Lumbosacral echinococcus may involve the pelvic cavity and the parasacral tissues, which leads to difficulty in clinical identification of the parasitic lesion. As in my series, involvement of the spine is frequently polyvertebral. In some cases, especially in its incipiency, it may be univertebral. Affected vertebrae are infiltrated and destroyed by the parasitic cyst. Not infrequently the pathologic process involves only the lamina; both spine and transverse processes, as well as the vertebral body, may be intact. In the majority of cases there is a microvesicular or multicystic appearance, with small sequestrums. Interestingly enough, the ligaments, cartilages and capsules are well preserved. Evidence of vertebral osteitis (spondylitis) is extremely rare, and there is usually no reactionary bone formation. Vertebral or spinal deformities appear very late in the disease and may produce a gibbus or axial deformity of the spine (the so-called hydatid spondylosis deformans). Hydatid pachymeningitis is extremely rare. The meninges are usually intact, especially the dura.

Pathogenesis.—Although still a matter of controversy, spinal echinococcosis is generally supposed to originate from the vertebrae, the paravertebral structures or the spinal cord itself. Irrespective of the primary location the parasitic lesion will invade and involve the surrounding structures and the spinal cord, causing vertebral deformities, resultant medaullary compression and organic neural manifestations.

Clinical Course and Symptoms.—The symptoms of echinococcosis of the spine simulate those of extramedullary spinal tumor. The infestation manifests itself in its early stages by radicular pain associated with objective sensory and motor disturbances. Reflex changes and amyotrophic alterations in the peripheral nervous system associated with local tenderness at the level of the involved vertebrae, not infrequently combined with progressive rigidity of the spine, may often be confused with tubercular spondylitis or Pott's dis-

ease unless extreme care is exercised. This is true particularly in those countries in which tuberculosis is a common disease. The most striking symptom, however, in the early stages of echinococcosis, are conservation of mobility, absence of deformity and rigidity of the spine, with sensory changes.

In my cases, despite the advanced lesions of the vertebrae, there were no apparent clinical signs of spinal deformity and no disturbances of mobility of the spine. In Case 1 the condition was thought to be tubercular spondylitis, and in Case 2 a spinal malignant tumor, by other members of the profession before the patients attended this Clinic.

Like other lesions involving or compressing the cord, as the echinococcus lesion advances organic neural changes appear distal to the site of the lesion. Paraplegia or quadriplegia with sensory and motor changes; sphincter disturbances; the Brown-Sequard syndrome; cauda equina, and conus syndrome are not infrequent. Intradural intramedullary spinal echinococcosis presents symptoms similar to those of central gliosis, syringomyelia or intramedullary tumors of the spinal cord. The foregoing symptoms are associated with median or paramedian painless tumor of the spine, which leads to the clinical diagnosis of spinal echinococcosis. Puncture of the cyst and microscopic examination of fluid confirms this diagnosis when the scolices of the parasite are identified.

Roentgen Examination.—Roentgen examination of the spine is important in the diagnosis of spinal echinococcosis. It reveals a rounded decalcified or punched-out area of unicystic or multicystic lesions. The intervertebral discs are usually well preserved, and there is no fusiform shadow of the spinal pattic abscess around the lesion. A myelogram is sometimes necessary and valuable to determine the exact location of the spinal block and to reveal the area of compression of the spinal cord.

Biologic Tests.—Spinal puncture, the Queckenstedt test and examination of the spinal fluid will reveal evidence of spinal block and confirm the diagnosis of impression of the spinal cord compression. Eosinophilia, the Weinberg and the intradermal reaction of Casoni are specific biologic signs of hydatidosis if they are positive. Unfortunately these tests frequently give negative, results, as occurred in my cases.

Fig. 1 (Case 1).—Roentgenogram showing lumbar kyphoscoliosis and advanced vertebral deformities.

Treatment.—Treatment of echinococcosis of the spine is unquestionably surgical and should not be delayed. To obtain good results it is essential to establish an early diagnosis. The prognosis is poor and recurrence to be expected if treatment is delayed. If an intradural intramedullary echinococcus cyst exists, a posterior midline myelotomy is necessary to remove it. Radical operation with complete removal is possible, and a quick cure can be expected. Injections of solution of formaldehyde into the cyst prior to operation and

formalinization of the region immediately after the operation has been considered a preventive measure to avoid a possible recurrence.

In my 1 case of echinococcosis of the lower lumbar portion of the spine the operation was so extensive that fusion of the spine was necessary. Fortunately this did not obtain in the case in which the cervical portion of the spine was involved. At the time of writing, the evidence points to a cure in both cases.

Fig. 2 (Case 1).—Anteroposterior (*A*) and lateral (*B*) encephalograms. Slight dilatation of left lateral ventricle is apparent (see text).

REPORT OF CASES

CASE 1.—N. F., a housewife 40 years of age, was admitted to the First Surgical University Clinic in February 1951, with combined paraplegia and right hemiplegia (triplegia), anesthesia below the umbilicus, sphincter disturbances and speech dysarthria, with emotional instability.

The history, obtained from relatives, revealed that the present illness had started with a bilateral sciatic pain and paresthesia of the lower extremities approximately three years prior to admission. The condition became gradually worse, and the patient was unable to walk because of paraplegia, with sensory loss and sphincter disturbances that developed at the end of second year, when she became totally bedfast. She was treated as a Pottic patient and was transferred to the Orthopedic Clinic for a checkup. While she was in the hospital there occurred a vascular cerebral accident, with aphasia and right hemiplegia. Operation was postponed on account of this unexpected complication, and the patient was discharged and taken home in this helpless condition.

Examination revealed a typical cauda equina syndrome combined with right hemiplegia, speech dysarthria and emotional instability due to a previous left cerebral vascular accident. The primary lesion was in the spine and spinal cord, as the history showed clearly. It involved the lower lumbar portion of the spine, causing a typical cauda equina syndrome with motor and sensory paralysis below the hip, sphincter disturbances and arriflexia. The cerebral accident, occurring later in the course of the spinal disease, complicated the picture.

Roentgen examination showed multiple microcystic and macrocystic lesions of the lower lumbar and upper sacral portions of the spine, characteristic of spinal hydatidosis. The lower portions of the third, fourth and fifth lumbar and the upper sacral vertebrae had been partially destroyed by the cystic lesions. There was an enormous geode between the third and fourth lumbar vertebrae. Lumbar kyphoscoliosis and advanced deformities of the vertebrae were apparent in the roentgenograms (Fig. 1).

A myelogram showed a definite block at the

third lumbar vertebra above the lesion.

Biologic tests (Casoni, Weinberg) gave negative results. There was no marked eosinophilia and no change in the blood picture and cell count.

An encephalogram showed slight dilatation of the left lateral ventricle, possible due to cerebral cortical atrophy, and intracranial traces of lipiodol in the base of the skull. There was no evidence of shifting or compression of the ventericular system. The spinal fluid was clear, and laboratory analysis revealed no abnormalities except moderate elevation in the protein content (0.75, Fig. 2).

In spite of the negative biologic reactions the clinical and roentgenologic signs strongly suggested spinal echinococcosis of the lumbosacral region, and the patient was taken to the operating room with this diagnosis on March 8. Operation was performed with the patient under general anesthesia (Kemithal, ether and oxygen). A midline incision was made in the lumbosacral region, and laminectomy was carried out in usual manner. Between the laminae of the fourth and fifth lumbar vertebrae small echinococcus cysts were protruding. The third, fourth and fifth lumbar laminae were removed; thousands of small echinococcus cysts with intercystic jelly-like substances were taken out; the area was carefully cleaned, and formaldehyde was applied. The gross specimen of these cystic lesions was exactly similar to ground beef (Fig. 3). The dura was opened, and no intradural extension or infiltration of the lesion was observed, but the dura was thickened in this region and the cauda equina greatly compressed.

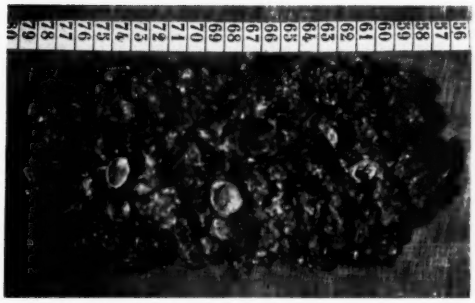


Fig. 3 (Case 1).—"Ground beef" appearance of gross specimen.

The fourth lumbar vertebra had been almost completely destroyed by the cystic lesion. This necessitated a bone graft and spinal fusion.

The postoperative course was long and the patient remained in the hospital for three months in a plaster cast, after which she was discharged to continue physical therapy for further improvement. At the time of her discharge she had regained control of the sphincters. There was marked improvement in the control of her legs.

CASE 2.—H. E., a farmer, 46 years old, was admitted to the Neurological University Clinic on Oct. 12, 1951, with a complaint of severe radiating pain in the arms and major weakness in the legs. His illness had started one year earlier with a dullness in the hands that gradually increased and was associated with radiating pain in both shoulders and arms. Three months prior to admission motor weak-

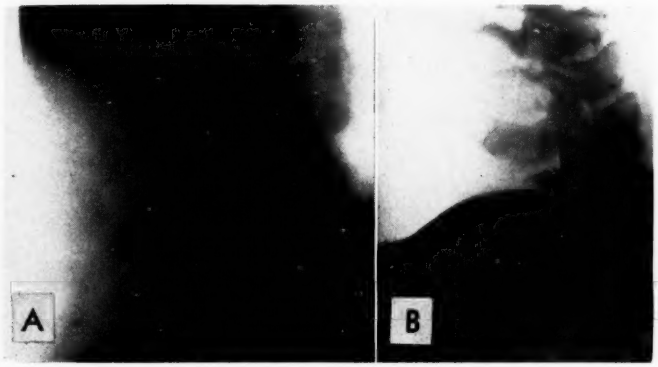


Fig. 4 (Case 2).—*A*, roentgenogram showing decalcification and partial destruction of second and fourth cervical laminal and spinous processes. *B*, myelogram showing complete block of contrast medium between second and third cervical segments.

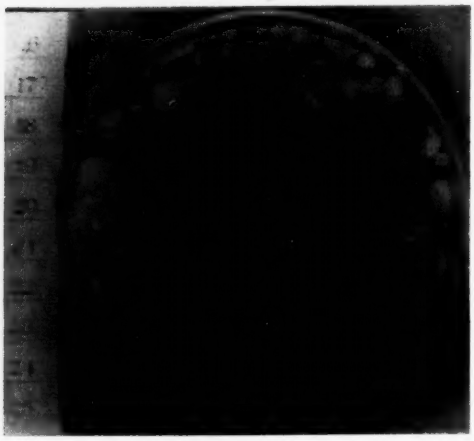


Fig. 5 (Case 2).—Echinococcus cysts packed in operative area, causing extradural compression of cord.

ness had developed in the upper and lower extremities, and walking became difficult. No sphincter disturbances occurred during the course of the illness. While the patient was in the Neurological Clinic his condition became increasingly and rapidly worse; he was soon confined to bed and unable to feed himself because of complete quadriplegia.

A tumor mass deep in the cervical region, extending to the right supraclavicular area, appeared and became palpable. Because of its consistency and immobility, the diagnosis of possible malignant tumor involving and infiltrating the upper cervical portion of the spine and resulting in spinal cord compression was made, and the patient was transferred to the First Surgical Clinic for operation on October 20.

Physical and neurologic examination revealed an upper cervical paramedian tumor, deep-seated and immobile, extending to the right supraclavicular region but causing no limitation of the mobility of the cervical position of the spine. There was no local tenderness to pressure and palpation. The tumor mass was hard and was about the size of an orange.

The patient was unable to walk or to feed himself because of spastic quadriplegia. Dynamometric examination of the power of the

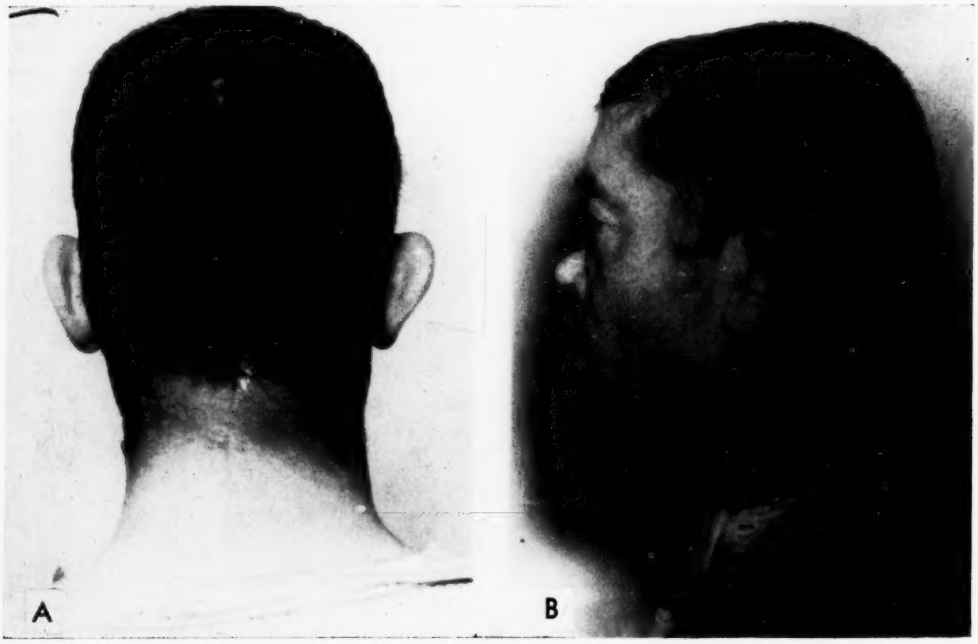


Fig. 6 (Case 2).—Posterior (*A*) and lateral (*B*) postoperative views of patient.

hands showed a decrease of 50 per cent in the right and 40 per cent in the left. Sensory examination revealed hypethesia and hypalgesia, with markedly decreased vibratory reception and deep sensation below the fifth cervical dermatome. A typical Horner's syndrome was visible on the right. The cutaneous reflexes were abolished bilaterally. The deep tendon reflexes were increased, clonus +, Babinskis were indifferent, Hoffman + bilaterally. There was no evidence of sphincter disturbances.

Repeated Wassermann and Kahn tests of both blood and cerebrospinal fluid gave negative results.

Spinal fluid: Clear — initial pressure 350 (Claude)
Lymphocytes 0.8
Protein 1.42
Chloride 7.94
Pendy +++
Nonne ++
Weichbrodt ++

Sedimentation
½ hr.—6 mm.
1 hr.—10 mm.
2 hr.—28 mm.

R.B.C. 4,000,000

W.B.C. 6,400

V.G. 84%. no eosinophilia

B. Colloidal—001122 222000

The Queckenstedt test showed complete block. Roentgen examination showed decalcification and partial destruction of the second and fourth cervical laminae and spinous processes (Fig. 4*A*).

A myelogram revealed complete block between the second and third cervical segments (Fig. 4*B*). The results of biologic tests were negative.

With the patient under general anesthesia (Kemithal, ether and oxygen) an upper cervical laminectomy was carried out in the usual manner. Between the spinal processes and the posterior arches of the second and third cervical vertebrae a typical enormous hydatid cyst was protruding. The laminae of the third and fourth cervical vertebrae, and part of that of the second, were removed and the echinococcus cysts carefully taken out. There were several hundred cysts of various sizes, packed in the area and causing spinal cord compression extradurally (Fig. 5). The dura was intact. A careful intradural exploration was made after the cysts had been completely removed. The region was formalinized, and the dura and the wound were closed in layers (Fig. 6).

The postoperative course was uneventful. The patient made a quick recovery, was up and about on the fifteenth postoperative day and was discharged one month after the operation, fully recovered.

Histologic examination of the surgical specimen confirmed the operative diagnosis. In this case the echinococcus cysts were entirely similar to the classic form of systemic echinococcus cysts and did not contain intercystic jelly-like substance as in the first case.

SUMMARY

Echinococcosis of the central nervous system, a comparatively rare condition, is discussed as to geographic incidence, pathogenesis, diagnosis and treatment. Six cases, in 4 of which the brain was affected and in the other 2 the spine and the spinal cord, were encountered by the author at the University Medical Clinic of Istanbul. Two cases are reported in detail.

Treatment, i. e., surgical intervention, should not be delayed. Good results require early diagnosis and prompt treatment. If operation is postponed, the prognosis is poor and recurrence is to be expected.

DISCUSSION BY DR. ROLAND KLEMME, ST. LOUIS

DR. KLEMME: I had the good fortune to visit Dr. Feyyaz Berkay's Clinic, and I was particularly impressed by the number of parasitic lesions encountered in Istanbul. Amebic abscesses of the liver and lung seemed extremely common. Solitary tuberculoma of the brain was very common, and Dr. Berkay was good enough to operate on several patients while I was there. We entered into a lengthy discussion on parasitic conditions of the central nervous system. As a result of these discussions I insisted that Dr. Berkay appear before this group and tell us some of his experiences.

In all my years of practice I have seen only

1 echinococcus cyst of the brain, in a child 9 years of age, with complete recovery. One other patient, 38 years old, had multiple parasitic cysts of the brain, first diagnosed as infectious granulomas, probably due to taenia mycosis. Later after further examination, it turned out to be due to a parasite that is fish-borne in the northwestern portion of the United States. The identity of the parasite was never established.

This presentation today is a most striking one. Our guest speaker in two and a half years has had the misfortune or good fortune, whichever you choose to call it, of seeing more parasitic cysts of the central nervous system than have been reported in his country in over fifteen years. The thing that is more impressive, however, is the value of sanitation, good horticulture and animal husbandry which we enjoy in this country, as compared to the countries in the Near East. Certainly, if any members of the audience are genuinely interested in seeing parasitic conditions and tuberculoma of the central nervous system, a visit to Dr. Berkay's Clinic in Istanbul would pay for itself many times over. Dr. Berkay's discussion is very timely in this day of potential global warfare. When our countrymen are scattered all over the world we should be better acquainted with common conditions in countries other than our own, in the event we are confronted with this type of clinical picture.

CLOSING REMARKS BY DR. BERKAY

DR. BERKAY: Basically, between intracranial and spinal echinococcosis there is no difference in the cysts. With spinal echinococcosis there is usually intercystic jelly or mastic substance, as in one of my cases. The intracranial echinococcus lesion is more commonly a unicystic or multicystic form that does not contain intercystic jelly. In the past two years I have had the opportunity to operate on 4 patients with cerebral echinococcus. In 3 of these the lesions were enormous single cysts; in only 1 of the 4 was multicystic hydatidosis of the brain observed. In this case I removed 105 small cysts from the frontal-parietal area of the brain. In these 4 cases the lesions were subcortically located, and 1 of them was a calcified hydatid cyst deeply seated in the parietal lobe. One of the most prominent symptoms of the cerebral echinococcus cyst is the convulsive seizure that was present in all my cases. All of the patients were surgically treated, with good results and no recurrences.

An outstanding characteristic of the spinal echinococcus cyst is that, in the majority of cases, the cysts are extramedullary and extradural. They originate primarily from the vertebrae or the paravertebral structures and involve the spinal cord secondarily. Intramedullary spinal echinococcus cysts are exceptional. Intracranial echinococcus cysts are more frequently primary intracerebral subcortical lesions, and those originating from the skull and involving the brain secondarily are rare, even exceptional.

ZUSAMMENFASSUNG

Die Echinokokkuserkrankung des Zentralnervensystems, eine verhältnismässig seltene Erkrankung, wird in Bezug auf geographische Verbreitung, Pathogenese, Diagnostik und Behandlung erörtert. Der Verfasser sah in der Medizinischen Universitätsklinik von Istanbul sechs Fälle, von denen vier die Erkrankung im Gehirn und die übrigen in der Wirbelsäule und im Rückenmark aufwiesen. Zwei Fälle werden im einzelnen dargestellt.

Die Behandlung, d. h. der chirurgische Eingriff, soll nicht verzögert werden. Gute Erfolge hängen von frühzeitiger Erkennung und sofortiger Behandlung ab. Aufschub der Operation verschlechtert die Prognose und mag zu Rückfällen Anlass geben.

RÉSUMÉ

On discute la pathologie géographique, la pathogénie, le diagnostic et le traitement de l'échinococcose du système nerveux central (rare). L'auteur décrit quatre cas d'échinoccose médullaire qu'il a observés à la Clinique universitaire de médecine d'Istamboul. Deux cas sont rapportés en détail. Traitement : l'intervention chirurgicale ne doit pas être différée. Seul un diagnostic précoce et un traitement rapide donnent de bons résultats. Si on retarde l'intervention, le pronostic est mauvais et on doit s'attendre à des récidives.

RESUMEN

La equinococcia del sistema nervioso central es un padecimiento raro. Se discute su incidencia geográfica, patogenia, diagnóstico y tratamiento. En la Clínica Médica Universitaria de Estanbul, el autor encontró 6 casos, de los cuales en 4 se encontraba afectado el cerebro y en 3 la médulla y la columna vertebral. Se comunican 2 casos en detalle.

El tratamiento ó sea la intervención quirúrgica no debe demorarse. Se requiere para la obtención de buenos resultados, diagnóstico precoz y tratamiento inmediato. Si se pospone la operación se enpobrece el pronóstico y es de esperarse la recidiva.

RIASSUNTO

E' un lavoro sull'echinococcosi del sistema nervoso centrale, malattia rara, di cui vengono studiate la distribuzione geografica, la patogenesi, la diagnosi, la cura. L'autore, nella Clinica Medica dell'Università di Istanbul, ne ha osservati 6 casi: in 4 era interessato il cervello, negli altri la colonna vertebrale ed il midollo spinale. Due casi vengono descritti dettagliatamente. La terapia è chirurgica, e non deve essere rimandata nel tempo. Per ottenere buoni risultati sono necessarie una diagnosi precoce ed una pronta terapia, altrimenti la prognosi è cattiva e possono verificarsi recidive.

REFERENCES

Benhamon, E., and Goinard: Echinococcose intrarachidienne Rev. Neurol. 2:657-697, 1929.

Dévé, F., and Griesel, P.: Rev. de Chir. Neurol. 6:375-463, 1929.

Yücell, F. A.: Hastane N. 1, 1953.

The notion that the physician or surgeon of today is wiser or safer than his forerunner simply because he has been dowered with a bewildering array of strange paraphernalia is to be not only emphatically denied, but militantly opposed. Unless he happens to be wise and careful by nature, the fruits of medical science are likely to make him all the more dangerous. The fruits of science are always spoken of as if they were invariably beneficent. They are no more so than the phenomena of nature. The water of the river is a phenomenon of nature; it will cleanse you or drown you with equal imperturbability.

—*Clendening*

Physiologic Mechanisms of Spinal Shock*

GEORGE M. AUSTIN, M.D.,
AND
GRAYSON P. McCOUCH, M.D., D.A.B.
PHILADELPHIA, PENNSYLVANIA

IT would seem imprudent for use at this time to review methodically the carefully documented work from 1833 to 1934 on spinal shock. Mere mention will be made to give credit to outstanding investigators on the way. Even the definition will bear little in the way of alteration, although we should like to mention it for its possible use in the course of discussion: a transient state of reflex depression occurring caudal to a lesion producing spinal transection. Certainly the condition of spinal shock *per se* deserves little in the way of study, for its foreboding presence is known only, with awe and alarm, to the clinician. However, as a mechanism causing temporary cessation of reflex activity and synaptic function with the gradual return and blending to a state of heightened activity, it is of extreme interest. It seems worthy of investigation, therefore, mainly as a pattern of action which, if understood, may facilitate insight into other patterns of action occurring at other levels to the nervous system. Eccles and McIntyre[1] define plasticity as the ability of individual synaptic junctions to respond to use and disuse by appropriate changes in transmissive efficacy. The mechanism of plasticity has been postulated by Konorski,[2] Penfield[3] and others as possibly underlying memory processes, and Eccles and McIntyre[1] have gone on to show that plasticity occurs even in the simplest of reflex arcs, namely, monosynaptic. It is not illogical, then, to carry over this concept to account for the eventual state of spasticity observed after spinal shock. Also, as others have noted, a period of cerebral shock frequently follows removal of large amounts of brain substance. This may be characterized by some loss of normal thought processes, memory and even consciousness. Yet to a remarkable extent, over a period of weeks, possibly through adaptation or plasticity, there is usually gradual return of function. To carry the analogy another step, we even have release mechanisms available on a cerebral basis, as after removal of an atrophic hemisphere,[4] that mimic to some extent the exaggeration of reflex activity of the forelegs observed in the Shiff-Sherrington phenomenon after spinal section. Thus, it seems to us that the mechanism and pattern of spinal shock are worthy of continued investigation in an effort to understand the delicate interplay that accounts for reflex diminution and accentuation and may explain similar phenomena at so-called higher levels of synaptic function.

Historical Summary. — Marshall Hall[5] is generally credited with first describing spinal shock in 1833. Not only did he describe it, but he made the observation that it is increasingly severe as one ascends the phylogenetic scale. Important contributions along the way were the description of the Shiff-Sherrington phenomena in 1898, and correction of the erroneous theories of Bastian[6] and Goltz[7] by Sherrington.[8] Bastian had confused the concept of surgical and spinal shock, theorizing that they were the same, whereas Goltz proposed the hypothesis that spinal

*Aided in part by a contract between the Office of Naval Research and the University of Pennsylvania and by the U. S. Public Health Service and the University of Pennsylvania.
From the Department of Neurosurgery (Dr. Austin) and the Department of Physiology (Dr. McCouch), University of Pennsylvania School of Medicine.
Read at the Eighteenth Annual Congress of the United States and Canadian Sections, International College of Surgeons, New York, Sept. 13-17, 1953.
Submitted for publication April 24, 1954.

shock was due to inhibition from descending impulses from the region of the transection.

In 1924 McCouch[9] demonstrated an increase in the flexion reflex following spinal transection on the side opposite that of previous motor cortex ablation, thus emphasizing the essential role of the pyramidal tract. Fulton, Liddell, and Rioch[10] in 1930 presented evidence showing that the vestibulospinal paths are largely responsible for the changes in the knee jerk following spinalization of the decerebrate animal. In 1940 Van Harevelд[11] advanced a theory that spinal shock actually depends on the dominance of a spinal inhibitory mechanism. This was based on the prompt onset of extensor rigidity after asphyxia following transection. This theory in turn is, of course, based on the fact that normal tone of the spinal cord is maintained by dominance of the downstream facilitative mechanism, which, as Sherrington showed, when obliterated by spinal transection completely releases the lower part of the cord from the influence of these higher centers. Hence, there is no great argument thus far among physiologists as to the actual condition prevailing at a cord level. As Sherrington has expressed it, there is a release of the segmental levels from the facilitatory action of the higher centers of the brain stem.

In addition, there is a strong spinal inhibitory mechanism, as shown by the increase in reflex excitability in the upper limbs when the cord is transected below the brachial enlargement. The precise site of spinal shock with regard to specific structures, as well as the mechanism of recovery, remains largely unknown. The brief definition given in an earlier paragraph fails, in many ways to describe the phenomena clearly. Moreover, it fails to account for, or to specify the duration of, areflexia and hyporeflexia. Also, it does not distinguish between a partial and a complete transection, or between an anatomic or a physiologic transection. However, it is now realized that in order to produce a stage of complete spinal shock it is not necessary to perform an anatomic transection. Under such conditions we describe this as physiologic transection, due to concussion or contusion of the cord.

From the human point of view, in high cervical physiologic transections occurring between the fifth and the sixth or the sixth and the seventh cervical vertebrae we have observed the following characteristics of high spinal transection: (a) depression of the pulse rate and, often, depression of respirations; (b) hyperthermia; (c) a drop in blood pressure; (d) complete loss of the autonomic reflexes, including those of the bladder and bowel; (e) slight plantar flexion of the great toe, elicited by stimulus on the plantar surface and occurring between four and eighteen hours after the injury;* (f) presence of the cremasteric reflexes or their recovery next to the plantar-flexor reflex; (g) recovery of the achilles tendon reflex in advance of recovery of the quadriceps reflexes, and (h) a tendency of the tendon reflexes to recover before the bowel and bladder reflexes had recovered.

Some of these data have been noted recently by others,[12] and it is merely to corroborate and reiterate the conditions associated with acute spinal shock in the human being that we mention them. In spite of disagreement over the mechanism and site of action of acute spinal shock, there are some points upon which there is generalized agreement. These are as follows:

1. There is a marked phylogenetic effect, so that generally the severity of spinal shock is increased as one ascends the evolutionary scale.
2. There is an initial period of reflex loss, measured in terms of seconds in lower forms and in terms of hours in man, with a gradual return of reflex function, eventually to a hyperactive stage.
3. There is both an earlier and a more

*This plantar reflex became fatigued very rapidly and it could only be elicited by a slight scratch over the flexor surface of the toe. Thus, it exhibited extreme local sign and seemed to be present when these patients were first seen.

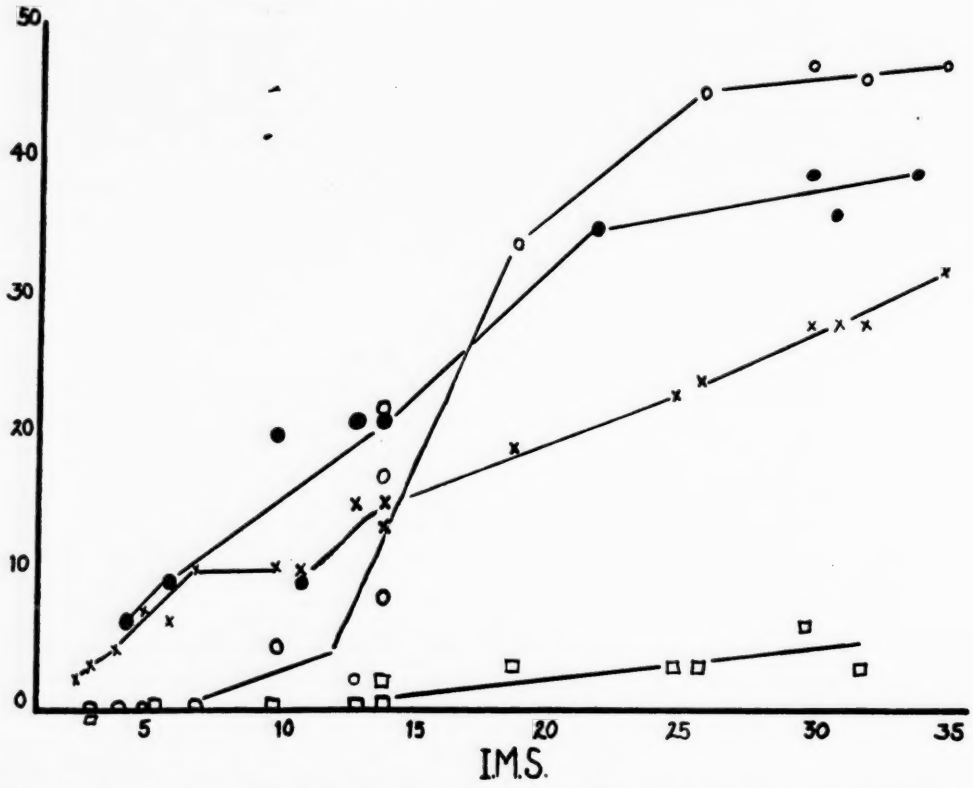


Fig. 1.—Decerebrate cat. Shows the development of the monosynaptic reflex (circle) the polysynaptic reflex (square) the dorsal root reflex (black dot) and the N_1 wave (×) of the negative intermediary potential with stimuli of increasing strength applied to the seventh lumbar dorsal root. The amplitude of these recorded factors is plotted on the ordinate against the amplitude of the intramedullary spike I.M.S. on the abscissa. Recording electrodes on the dorsum of the cord and on the seventh lumbar ventral root.

complete recovery of reflexes in lower forms, often sufficient to permit ambulation of different degrees.

4. In mammals the flexor reflex recovers first and eventually becomes exaggerated. Likewise, there is a tendency to recovery of the peripheral reflexes in advance of the proximal.
5. Generally the recovery of somatic reflexes precedes that of autonomic reflexes.
6. There is a much earlier and more complete recovery of function and reflex activity in immature animals.
7. In a progressive type of spinal transection in man, that is, one occurring over a period of some two weeks, there is no true spinal shock but a gradual transition to spasticity.
8. During recovery in mammals there is a gradual transition from hyporeflexia to hyperreflexia.
9. After recovery from spinal shock there is no secondary spinal shock if the cord is again transected just below the level of the original lesion.

Previous work by McCouch, Stewart, and Hughes[13] had shown that changes occur in both the negative intermediary potential and the positive intermediary potential in the presence of acute spinal shock. In the opinion of these authors,

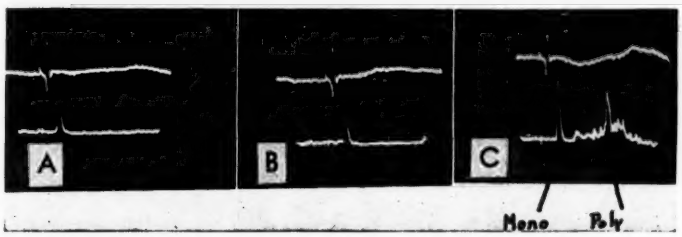


Fig. 2.—Records of cat in acute spinal shock (Jan. 2, 1953): stimulus, 0.8 volt to nerve from gastrocnemius. Upper record, dorsum of cord; lower record, seventh lumbar ventral root. *A* and *B*, decerebrate; *B* after curare, *C* after transection of cord.

they had demonstrated that the site of action in the presence of spinal shock was predominantly internuncial in the cat, while in the monkey there was in addition to internuncial depression, some shocklike effect on cells in the ventral horn. Because of improved instrumentation and technics, they concluded that further study of spinal cord and ventral root potentials might yield additional information concerning the mechanism of spinal shock.

Methods.—Decerebrate cats and monkeys were studied before and after acute midthoracic spinal transection. All animals were decerebrated under ether and allowed a minimal period of one hour before recording was begun which has previously been shown to be adequate.[14] Spinal transection was carried out with the animal under divinyl ether anesthesia, or, more frequently, under anesthesia induced by small intravenous doses of D-tubocurarine, 0.22 mg. per kilogram of body weight. The dose of D-tubocurarine was small enough so that control cord and ventral root potentials taken before and after its administration showed no changes.

Routinely, the lumbosacral cord was exposed and an appropriate dorsal root or nerve selected for stimulation. Potentials were then recorded from the surface of the spinal cord, the dorsal root and/or the ventral root. The dura was opened under warm Ringer's solution, and the cord and roots thereafter covered with warm mineral oil. Surgical shock was prevented by the administration of 40 to 80 cc. of intraperitoneal Ringer's solution.

Results and Comment.—Prior to transection in the decerebrate animal, the monosynaptic response appeared at threshold whether a muscle nerve, a mixed nerve, or a dorsal root was stimulated. A small polysynaptic response often appeared as one approached maximum stimulation strength. Also, it is interesting to note that a small N_1 wave was always

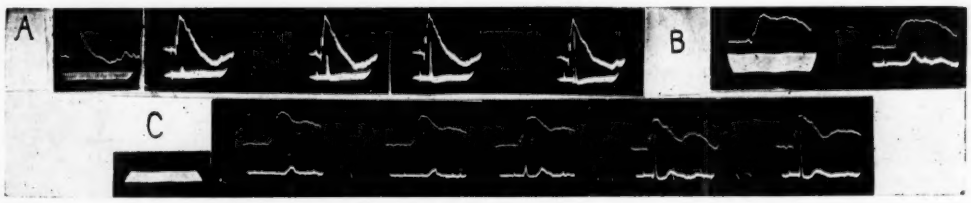


Fig. 3.—Baby macaca irus (Jan. 9, 1953). Stimulus to peroneal nerve. *A*, decerebrate; at volts 1, 0.4, 0.6, 0.8, 1; seventh lumbar ventral root recorded. *B*, spinal; at volts 1.4; seventh lumbar ventral root recorded. *C*, spinal; at volts 0.8, 1, 1.4, 1.8, 2; sixth lumbar ventral root recorded. Time, 1,140 cycles. The top sweep records the cord potential and the bottom sweep the ventral root potential in each record. In row *A* it is noteworthy that there is little if any polysynaptic response, while in row *C*, after spinal transection at midthoracic level, there is now a large polysynaptic component which is the threshold response.

present in the cord potential with the onset of polysynaptic activity.

The polysynaptic response and the negative intermediary potential (NIP) were larger if the stimulation was applied to a mixed nerve or a dorsal root. After spinal transection, however, the polysynaptic response was either the threshold response or appeared simultaneously with the monosynaptic. Again this reflex was associated with a negative intermediary potential that remained smallest in the case of the muscle nerves.

Figure 1 shows the growth of monosynaptic and polysynaptic reflexes plotted against height of the intramedullary spike (IMS) in the decerebrate cat. In addition, we also show the dorsal root reflex and N_1 developing as the amplitude of the intramedullary spike increases in response to stimulation of the seventh lumbar dorsal root.

Figure 2 illustrates the change in response from monosynaptic to polysynaptic following spinal transection as one stimulates the nerve to the gastrocnemius muscle. This figure shows that the minimal dose of curare used for immobilization does not alter the cord or ventral root potential. It also shows that stimulating a muscle nerve yields a poor N_1 potential even after the appearance of the polysynaptic reflex. The change in threshold response from monosynaptic to polysynaptic is displayed in Figure 3. There has been a slight change in position of the electrode on the surface of the cord at the time of spinalization, and this accounts for the decrease in amplitude of the intramedullary spike.

When facilitation and inhibition were tested before and after spinalization a marked change occurred. This is shown where the monosynaptic response from

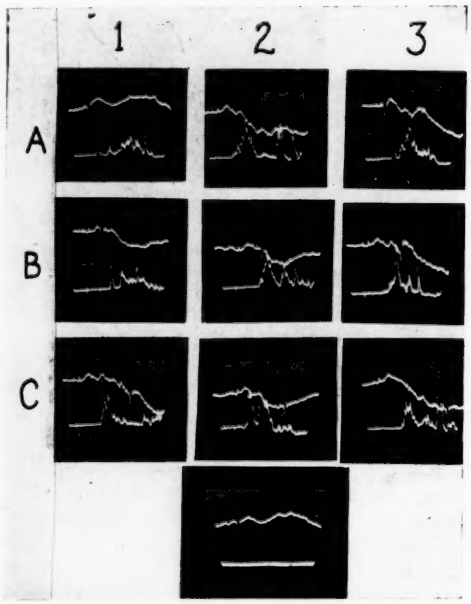


Fig. 5.—Conditioning effect of internal saphenous on gastrocnemius after acute spinal transection. The effect is the reverse of that observed in decerebration. In *A1* is the test response and in *A2* the conditioning response. *A3* shows early facilitation, but all subsequent pictures show inhibition of the monosynaptic test reflex at increasing separate distances.

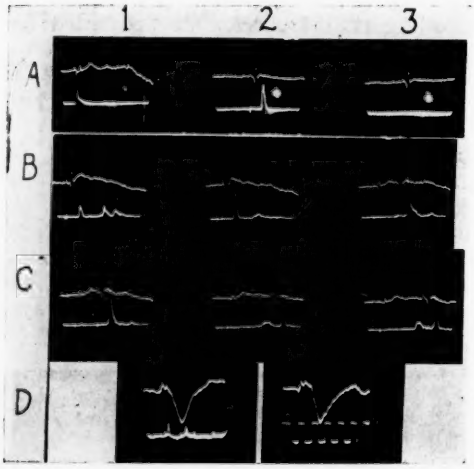


Fig. 4.—Decerebrate cat. Test response is shown in *A2*. Top sweep is cord potential and bottom sweep is ventral root potential following stimulation of gastrocnemius nerve and recording from seventh lumbar segment. Conditioning response is from stimulation of internal saphenous nerve and is shown as recorded from the *dorsal root* in *A1*. Time is 1,140 cycles. Rows *B* and *C* show the conditioned response of the nerves at increasing times and after *B3* facilitated. Row *D* shows the cord response at a slow sweep (60 cycles) from stimulation of the internal saphenous nerve.

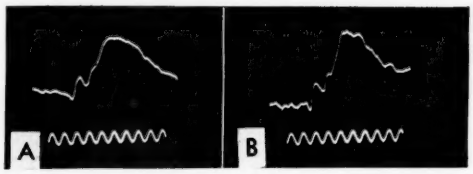


Fig. 6.—Effect of spinalization on the negative intermediary potential, with increased discreteness of aN_1. Baby macaca irus (Jan. 7, 1953) curarized; stimulus to peroneal nerve. *A*, decerebrate; *B*, four minutes after transection of cord.

the gastrocnemius nerve is conditioned by a volley from the internal saphenous nerve in both the decerebrate and the spinal state. Figure 4 shows that, in the decerebrate cat, the monosynaptic reflex from the gastrocnemius is inhibited for the first few milliseconds and thereafter strongly facilitated for at least thirty to forty milliseconds. After spinalization (Fig. 5) the effect is reversed, there being an initial facilitation for a few milliseconds and thereafter a rather prolonged depression or inhibition of the monosynaptic response. These results appear to be in keeping with the general observations made on animals, namely, that in the decerebrate state there tends to be an exaggeration of the extensor monosynaptic response (stretch reflex) and in the spinal state a depression thereof.

The alterations in the N_1 part of the cord potential are more pronounced in the monkey than in the cat after spinalization, as is, of course, spinal shock. Briefly, the changes we have observed in the negative intermediary potential following spinalization of the decerebrate and unanesthetized animal are as follows:

1. Prolongation of the negative intermediary potential, with rounding out of N_1 and N_2; generally this change seems best described by the term "desynchronization." It is somewhat transient and is more marked in the monkey than in the cat.
2. A transient depression of the positive intermediary potential, or so-called P wave. This is also more marked in the monkey.
3. The appearance, or an increase in the discreteness, of a small wave on the rising phase of N_1, hereafter called aN_1.

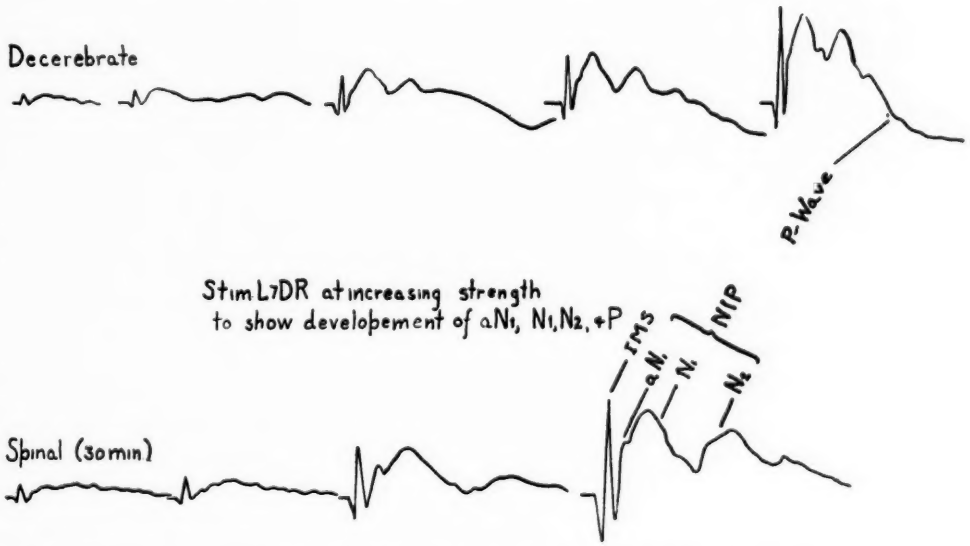


Fig. 7.—Development of the cord potential with increasing strength of stimuli, before and after acute spinal section.

The so-called desynchronization of the negative intermediary potential and the emphasis of a separate wave, aN_1, on the rising phase of N_1 is seen in Figure 6. This is shown in a series of stimuli of increasing strength in Figure 7. These records were obtained by tracing the separate projected negatives.

From these results obtained after spinalization it seemed to us that aN_1 and the remainder of N_1 proper could represent separate pools of interneuronal response. However, subsequent data obtained from asphyxial and conditioning experiments have shown that aN_1 behaves in a fashion similar to the intramedullary spike and unlike the remainder of N_1.[15] For this reason we have chosen to interpret aN_1 as presynaptic. This suggests that one of the effects of desynchronization is to fractionate the negative intermediary potential with presynaptic and postsynaptic components.

SUMMARY AND CONCLUSIONS

1. The historical development of the concept of spinal shock is reviewed.
2. The possible significance of the theory of reflex loss with gradual return to hyper-reflexia is considered relevant to synaptic failure and return of function at higher neural levels.
3. The cardinal physiologic observations after high cervical transection in man are enumerated.
4. The results of recent investigations on spinal shock in the cat and the monkey suggest the following conclusions:
   a. There is a shift of emphasis from the monosynaptic reflex in the decerebrate animal to a threshold polysynaptic reflex in the spinal animal.
   b. After spinal transection there is a desynchronization of the negative intermediary potential of the spinal cord with a tendency to fractionate this potential into an early presynaptic and a later postsynaptic component.

DISCUSSION BY DR. J. LAWRENCE POOL, NEW YORK

DR. POOL: I have been most impressed by the physiologic studies just reported, which in my opinion mark a great step toward understanding of the mechanisms of spinal shock. Indeed, this is about the only way in which we can hope to understand the physiologic disturbances in the spinal cord after injury or disease resulting in a transection. It does not seem to me, however, that the animal studies reported necessarily duplicate the condition in man during the period of spinal shock characterized by areflexia and flaccid paralysis that usually lasts several weeks after transection of the human spinal cord.

The studies of electrical changes in the cat and monkey cord after transection indicate enhancement of the polysynaptic responses. This suggests a state that would more probably resemble the condition in man when the period of "spinal shock" has ended and hyperreflexia, with involuntary or "spastic" movements, supervenes.

I should therefore like to ask the speakers whether they correlate their results with the clinical state of true spinal shock in man, or with the later state of hyperreflexia and "spasticity" that usually follows spinal shock.

DISCUSSION BY DR. CHANDLER MCBROOKS, BROOKLYN

DR. MCBROOKS: In discussing this paper on the mechanism of spinal shock it is pertinent to point out that all general body reactions are due to an interplay between higher and lower integrative centers.

It is an orthodox physiologic concept that these lower centers have acquired so much dependence upon directives from above that, when connections with higher centers are cut off, total inactivity or spinal shock results for a time.

It can also be said that there are two types of integrating or spinal response mechanisms: the two-neuron arc or monosynaptic reflex pathway, and the polysynaptic pathway. Higher centers act upon both. What is the peculiar functional significance of each?

The monosynaptic reflex is in integral part of the antigravity system. It, however, seems to have a modulator action, whereas the polysynaptic pathways are essential to purposive, effective or complex responses. These monosynaptic reflex responses are initiated by activity in progress, such as stretch imposed on muscle by gravity, and they break through

other activities to check or reenforce a movement.

In some ways it is surprising that Dr. Austin has noted lower thresholds for and dominance of the polysynaptic reflex after cord section. The monosynaptic pathway characteristically is less susceptible to block by anesthetics, by such drugs as myanesin, by asphyxia, etc. As a matter of fact, the reflex responses to tapping of tendons, which are among the early signs of recovery from spinal shock, are monosynaptic responses. In my opinion, any lessening of prominence of the monosynaptic reflex in spinal animals is due to (1) absence of facilitative action of higher "antigravity centers" and/or (2) overaction of the polysynaptic reflexes caused early by irritation from the site of cord section and later by release of these polysynaptic complexes from higher integrative control. A high background of polysynaptic activity has an inhibitory action on the monosynaptic pathway. This question of relative dependence of the two types of reflex on higher centers is of interest and deserves continued study.

Dr. Austin is to be commended for his effort to determine what is actually happening in the spinal cord during reflex activity. This is a difficult procedure. Interpretation of the complexities of cord potentials is one of the standard methods of obtaining information. One of the major problems is to determine whether a material or a condition acts upon terminals of an axon at a synapse or upon the neuron somas near or on which they terminate. His argument that the aN_1 potential is given by the afferent terminals and is less affected by asphyxia than the N_1 potential is interesting but raises questions that should be discussed at greater leisure. There is evidence that the terminals, owing to their greater surface area, have a higher oxygen requirement and are more susceptible to asphyxia than are the motor neurons as indicated by antidromically induced soma and dendritic potentials. Since in Dr. Austin's work the aN_1 potential was reduced by asphyxia at the same time that the initial negative (N_1) potential was abolished, while a residual component of aN_1 remained, as did fiber potentials, I suggest that this aN_1 component is really a complex picked up from the afferent fiber and the terminals. As asphyxia occurs, the terminals are blocked, which result in failure of the interneuron stimulation and the absence of N^1. A residue of aN_1 remains because, like other fibers, the main intracord segments of the activated afferents are not readily blocked — they contribute the unimpaired component of the aN_1 potential.

ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNGEN

1. Es wird ein Überblick über die geschichtliche Entwicklung der Auffassung vom Rückenmarkschock gegeben.

2. Es wird angenommen, dass die Bedeutung der Theorie des Reflexverlustes mit allmählicher Rückwandlung in Hyperreflexie möglicherweise in ihrer Anwendbarkeit auf das Versagen der Synapsis und auf die Wiederaufnahme der Funktion durch höher gelegene Nervenzentren beruht.

3. Die hauptsächlichen physiologischen Beobachtungen nach hoher Durchschneidung des Halsmarks beim Menschen werden aufgezählt.

4. Die Ergebnisse neuer Untersuchungen des Rückenmarkschocks bei Katzen und Affen lassen die folgenden Schlussfolgerungen zu:

(a) Die Einschätzung der Bedeutung des monosynaptischen Reflexes beim enthirnten Tier hat sich zugunsten eines polysynaptischen Schwellenreflexes beim Rückenmarkstier verschoben.

(b) Nach Durchschneidung des Rückenmarks kommt es zu einer Desynchronisierung des negativen intermediären Potentials des Rückenmarks mit Neigung zur Fraktionierung dieses Potentials in eine frühe praesynaptische und eine spätere postsynaptische Komponente.

RESUMEN

1. Se revisa el desarrollo histórico del concepto de choque espinal.

2. La posible significación de la teoría de la pérdida de los reflejos con vuelta gradual a la hipereflexia se considera apropiada a la falla sináptica y vuelta funcional a niveles nerviosos superiores.

3. Se enumeran las observaciones fisiológicas cardinales después de la sección medular cervical alta en el hombre.

4. Los resultados de investigaciones recientes en el choque espinal en el gato y en el mono sugieren las conclusiones siguientes:

a. Existe un cambio a enfatizar desde el refeljo monosináptico en el animal decerebrado hasta el reflejo polisináptico umbral en el animal espinal.

b. Después de la sección espinal existe una desincronización del potencial negativo intermedio de la médula espinal con una tendencia a fraccionar su potencial en un componente primario presináptico y uno tardío postsináptico.

CONCLUSIONI RIASSUNTIVE

1. Viene passato in rassegna lo sviluppo storico del concetto di shock spinale.

2. Il possibile significato della teoria della perdita dei riflessi con graduale ritorno alla iper-reflessia viene considerato in relazione alla perdita della sinapsi e al ritorno di una funzionalità a livelli neurali più alti.

3. Vengono enumerate le principali osservazioni dopo interventi sul midollo cervicale nell'uomo.

4. I risultati dei recenti studi sullo shock spinale nel gatto e nella scimmia portano alle seguenti conclusioni:

a) esiste un mutamento di ampiezza fra i riflessi monosinaptici nell'animale decerebrato e i riflessi polisinaptici nell'animale spinale;

b) dopo resezione spinale si instaura una desincronizzazione del potenziale intermediario negativo del midollo spinale con tendenza a frazionarsi in due componenti: pre-sinaptica precoce e post-sinaptica tardiva.

REFERENCES

1. Eccles, J. C., and McIntyre, A. K.: Plasticity of Mammalian Monosynaptic Reflexes, Nature 167: 466-468, 1951.

2. Konorski, J.: Conditioned Reflexes and Neuron Organization. Cambridge, Mass.: Cambridge University Press, 1948.

3. Penfield, W.: Memory Mechanisms, Arch. Neurol. & Psychiat. 67:178-191, 1952.

4. Penfield, W.: Ablation of Abnormal Cortex in Cerebral Palsy, J. Neurol., Neurosurg. & Psychiat. 15:73-78, 1952.

5. Hall, M.: New Memoirs on the Nervous System. London: H. Bailliere, 1843. Fulton, J. F.: Physiology of the Nervous System. Oxford University Press, 1949.

6. Bastian, C.: On the Symptomatology of Total Transverse Lesions of the Spinal Cord, Med. Chir. Trans. 73:151, 1890.

7. Goltz, F.: Ueber die Functionen des Lendenmarks des Hunds, Pfliig. Arch. ges. Physiol. 8: 460-498, 1874.

8. Sherrington, C. S.: Experiments in Examination of the Pedipheral Distribution of the Fibers of the Posterior Roots of Some Spinal Nerves, Phil Trans. Roy. Soc., Part II, 1897, 190B:45-186.

9. McCouch, G. P.: The Relation of the Pyramidal Tract to Spinal Shock, Am. J. Physiol. 71: 137-152, 1924.

10. Fulton, J. F.; Liddell, E. G. T., and Rioch, D. McK.: The Influence of Unilateral Destruction of the Vestibular Nuclei Upon Posture and the Knee Jerk, Brain 53:327-343, 1930.

11. van Harreveld, A.: On Spinal Shock, Am. J. Physiol. 129:515-523, 1940.

12. Poole, J. L.: The Neurological Treatment of Traumatic Paraplegia. Springfield, Ill.: Charles C Thomas, Publisher, 1951.

13. McCouch, G. P.; Stewart, W. B., and Hughes, J.: Cord Potentials in Spinal Shock Single Volleys, J. Neurophysiol. 3:139-145, 1940. Cord Potentials in Spinal Shock Multiple Stimuli, J. Neurophysiol. 3:146-150, 1940. Cord Potentials in Spinal Shock Crossed Effects in Monkey, Macaca Mulatta, J. Neurophysiol. 3:151-155, 1940.

14. Austin, G. M., and Pask, E. A.: Effects of Measured Blood Ether Concentrations on Monosynaptic and Polysynaptic Cord Reflexes, J. Physiol. 118:405-411, 1952.

15. Austin, G. M., and McCouch, G. P.: Fractionation of the Negative Intermediary Potential of the Spinal Cord into Presynaptic and Postsynaptic Components, to be published.

Relief of Spasticity in Paraplegia

IRVING S. COOPER, M.D.

NEW YORK CITY, NEW YORK

ONE of the most distressing and incapacitating neurologic sequelae of injury to the spinal cord is intractable spasticity of the paralyzed extremities. In persons with traumatic paraplegia, spasticity of the paralyzed lower extremities becomes marked enough to impede rehabilitative measures in approximately 40 per cent of those with cervical and thoracic cord injuries. The relief of this spasticity will often enable a bedridden paraplegic to become ambulatory and self-sufficient. It is the purpose of this report to outline the neurosurgical methods that will alleviate intractable spasticity in the paraplegic patient.

Physiologic Aspects of Spasticity.—As the isolated spinal cord recovers from spinal shock, the first reflex to be elicited usually is a dorsiflexion withdrawal of the foot or great toe when the sole is stimulated. Gradually the segmental and more complex intersegmental reflexes return and enter a stage of heightened reflex activity.

Flexor Response: In the majority of cases, reflex activity of the isolated cord is predominantly flexor. Simple dorsiflexion of great toe and foot on stimulation of the sole will later spread to involve the hamstring and iliopsoas muscles, resulting in simultaneous flexion withdrawal of foot, knee and hip. Eventually, flexion of the stimulated extremity may be accompanied by extension or adduction, or both, of the opposite lower limb, owing to overflow of afferent impulses into intersegmental and commissural neuron pools. The deep tendon reflexes, as well as the cremasteric and bulbocavernosus reflexes, are attainable during this phase of reflex activity. In an occasional case "reflex stepping" may be elicited at this time; that is, with the knee flexed, sudden gentle dorsiflexion of the foot causes extension of the ipsilateral knee and ankle accompanied by flexion of the contralateral knee and ankle. In some instances only the ipsilateral extension occurs, in which case the reflex is referred to as "extensor thrust." The lower extremities are at rest in an attitude of moderate flexion in subjects who manifest the flexor response.

Extensor Response: In a minority of cases the paralyzed limbs of "spinal" man become fixed in extensor, rather than flexor, rigidity. In such instances the hips and knees are extended, with the feet and toes plantar flexed. Stimulation of the sole results in exaggeration of extensor response. Although deep tendon reflexes are only slightly or not at all elicitable, clonus is demonstrable at knee and ankle. Flexion is predominantly a defensive spinal reflex, whereas extension is a postural reflex dependant upon the brain stem and the midbrain centers. It has been postulated that, in cases of incomplete transection of the cord in which there is extensor rigidity, reticulospinal and vestibulospinal pathways mediate the extensor response. It is established, however, that extensor spasticity may occur in the presence of a complete cord transection.

The Mass Reflex: In approximately 50 per cent of cases in which the flexor response is demonstrated during the phase of heightened reflex activity the reflexes become increasingly exaggerated, gradually responding to stimulation of the sole as a "mass reflex." At this time a relatively minor stimulus results in a diffuse outburst of motor energy from all segments of the isolated cord. This response is characterized by acute flexion at the

Read at the Eighteenth Annual Congress, of the United States and Canadian Sections, International College of Surgeons, New York, Sept. 13-17, 1953.
Submitted for publication July 13, 1953.

ankles, knees and hips, with spasm of the abdominal musculature, particularly the recti, below the level of injury to the cord. In addition there is overflow of the nervous energy into the lateral columns, resulting in an autonomic outburst characterized by profuse sweating. There is also evacuation of the bowel and bladder. This "mass reflex" is a powerful defensive spinal reaction and is characteristic of the activity of spinal reflex arcs isolated from suprasegmental inhibitory influences. In addition to this "release" mechanism, Scarff and Pool have presented evidence of an "irritative mechanism" produced by scarring of the distal stump of the cord, which also contributes to the development of the mass reflex in man.

As the mass reflex becomes increasingly intense, the area for reception of afferent stimuli capable of calling forth this reflex *en masse* becomes more widespread and more readily receptive of exceedingly minor stimuli. Eventually, stimulation of any point on the paralyzed limbs may produce the mass reflex, and at times it is occasioned by visceral stimulation.

The clinical sequelae of such hyperreflexic phenomena are well known. Constant reflex movements of the lower extremities and the abdominal musculature contribute to formation of decubitus ulcers, discourage adequate intake of food, often produce vomiting, prevent development of useful automatic bladder function, and prevent rehabilitation. Fortunately, neurosurgical relief of the most intractable spasticity is not technically difficult or dangerous. One must accept the fact that satisfactory relief of spasticity depends upon the sacrifice of certain nervous elements which are producing involuntary muscular contraction. However, since these elements will never produce useful motor power, the only significance of the sacrifice is the realization it brings to the patient that these motor nerves are permanently gone. The ultimate benefits of such a realistic approach will prove immeasurable.

Technical Procedures.—The procedures to be described can be classified into four types: (1) peripheral neurectomy, (2) subarachnoid alcohol block, (3) rhizotomy and (4) subtotal spinal cordectomy.

The resection of peripheral nerves has rather limited usefulness in relief of spasticity in paraplegic persons. It is confined largely to resection of the obturator nerves for relief of adductor or scissors spasticity. When the incapacitating spasticity is largely confined to the adductor muscles of the thighs, the procedure of choice is bilateral transabdominal obturator neurectomy. This procedure can be performed easily and provides immediate relief. The nerve is approached retroperitoneally through a McBurney abdominal incision, and is readily identified as it emerges from the medial side of the psoas muscle to cross the brim of the pelvis. Stimulation of the nerve to produce adduction of the thighs positively identifies it prior to resection.

Subarachnoid alcohol block has emerged as the single most useful and most applicable method available for relief of paraplegic spasticity. The indications for subarachnoid alcohol injection are essentially those previously applied to anterior rhizotomy. The chief indication is intractable spasticity of the lower extremities, which have no useful motor or sensory function. Enough time should have elapsed from the time of injury so that the lesion may be known to be stationary; this eliminates the possibility of future neurologic recovery. This procedure has certain advantages over anterior rhizotomy. It is safer and more easily performed and is free of the hazards of major surgical procedure. Its effects are immediate and, in most instances, of long duration. It is, however, not selective. Thus, if certain nerve roots, such as those to the detrusor urinae, are to be excluded, this method is contraindicated in favor of selective anterior rhizotomy. Such contraindications, however, are rare, particularly since it has been demonstrated that satisfactory automatic micturition and sexual potency in the male can develop after subarachnoid alcohol injection.

The technic of subarachnoid injection

I now use is as follows: The patient is placed on his side in bed, and the foot of the bed is elevated 18 inches. An 18-gauge spinal puncture needle is inserted at the first lumbar interspace. A syringe containing 15 cc. of absolute alcohol is attached to the needle, and the alcohol is injected at the rate of 1 cc. every fifteen seconds. An assistant constantly checks the patient's vital signs and the level of anesthesia during the injection. The injection is continued until both legs have become completely flaccid or until 15 cc. of alcohol has been injected. The patient is then turned on his back and kept in this position for four to six hours. He is allowed out of bed on the following day. If the injection has not produced satisfactory flaccidity, it is repeated the following day, an additional 10 cc. of alcohol being used.

Relief of spasticity has been immediate and complete in 46 consecutive cases in which I have used this procedure. However, I have had 4 patients in whom spasticity returned within six to eighteen months. Satisfactory results were not obtained in these 4 patients by secondary alcohol injection, and surgical measures were resorted to. In 2 cases of quadriplegia, relief of spasticity in the lower extremities by alcohol injection was followed by onset of severe spasticity in the abdominal myotomes immediate superior to the level of the block. This subsided spontaneously within four weeks in each case. There have been no serious sequelae and no deaths in my personal experience with subarachnoid alcohol injection for paraplegic patients.

Rhizotomy, or intraspinal resection of nerve roots, is the time-honored method of relieving paraplegic spasticity since the introduction by Foerster of posterior root section for spastic paraplegia. The fact that posterior rhizotomy alone did not provide adequate relief of spasticity led Munro to develop the technic of anterior dorsolumbar rhizotomy. This method requires laminectomy and division of all anterior nerve roots from the tenth thoracic through the first or second sacral. The last firm dentate ligament is usually regarded as indicating the level of the first lumbar nerve root, although this relation is not invariable. If one is anxious to avoid sacrificing the motor supply of the detrusor urinae (usually second and third sacral), these roots can be positively identified by stimulating each sacral root individually and at the same time recording the intravesical pressure. Unless one is desirous of sparing certain nerve roots, such as those which innervate the urinary bladder, or unless some sensation exists in the lower extremities, subarachnoid alcohol injection is probably to be preferred to anterior rhizotomy. Kuhn has stated that subarachnoid alcohol block should be restricted to patients with total lack of detrusor innervation as determined by cystometric studies. However, it is now apparent that this criterion is too rigid, since satisfactory reflex emptying of the bladder is frequently achieved after subarachnoid alcohol block. Consequently, the indications for anterior rhizotomy have decreased markedly, and this operation is seldom used in my own paraplegic service.

Occasionally one is faced with the problem of recurrent spasticity following subarachnoid alcohol injection. In these circumstances the operation of anterior rhizotomy is made very difficult by the dense adhesive arachnoiditis produced by the prior alcohol injection. This arachnoiditis usually makes positive identication of individual roots impossible. In 4 such cases I have resorted to subtotal resection of the spinal cord. This subtotal cordectomy involves resection of the spinal cord and nerve roots below the level of the ninth or tenth thoracic segment. The operation requires a laminectomy extending from the ninth or tenth thoracic to the first lumbar level and is performed more quickly and easily than is anterior dorsolumbar rhizotomy. I have performed subtotal spinal cordectomy for intractable spasticity in 4 paraplegic patients.

McCarty has demonstrated that removal of the entire cord in man below the second thoracic level is not incompatible with life. In my limited experience, removal

of that section of the spinal cord below the ninth thoracic level has had no untoward or unexpected sequelae. All 4 patients in this group have demonstrated return of useful contractions of the urinary bladder, and the first of them has been discharged with a satisfactory automatic bladder. One patient in this group has been relieved of intractable pain in the lower extremities, as well as spasticity, after subtotal cordectomy. There is, of course, no possibility that recurrent spasticity will follow this procedure, and subtotal cordectomy deserves further evaluation in the treatment of intractable pain and spasticity in paraplegic patients.

SUMMARY

The physiologic background, clinical manifestations and neurosurgical treatment of spasticity in paraplegia are reviewed. Spasticity in paraplegia requires neurosurgical intervention when it renders adequate rehabilitation impossible, has not responded to conservative management, and involves muscles that have no useful voluntarily controlled function. The principal neurosurgical methods for relief of spasticity are peripheral neurectomy, subarachnoid injection of alcohol, selective anterior rhizotomy and subtotal spinal cordectomy. The proper method, judiciously selected and applied, will invariably result in alleviation of spasticity and improvement in the care of the paraplegic patient.

DISCUSSION

BY DR. O. H. FULCHER, WASHINGTON, D. C.

DR. FULCHER: I wish to thank you for permitting me to participate on the program. I had not read Dr. Cooper's paper previously, but I am very much impressed by his use of subtotal cordectomy for patients suffering with spastic paraplegia. I should like to confine my remarks to this facet of his splendid essay.

I was a surgeon from 1933 to 1937 in an industrial community where there occurred many back injuries, usually as a result of a man's being "jack-knifed." He would be working in a thin vein of coal which would require his remaining in a partially flexed position. Sometimes a large boulder would drop on his shoulders, which would cause extreme flexion of the body on the thighs. Almost invariably there would result a compressed fracture of the body of the twelfth dorsal or first lumbar vertebra. These patients invariably suffered with a lower motor neuron type of paralysis of the lower extremities. Hence there developed no spasm of the muscles of the lower extremities. In patients with a compressed fracture of the body of the eleventh dorsal vertebra, with resulting paralysis of the lower extremities spastic paraplegia and massive reflex movements did frequently develop. Thus it was concluded by clinical observation that a physiologic transection of the spinal cord at the twelfth dorsal vertebra represented the highest level at which the consistent result would be flaccid paralysis of the lower extremities. As a rule it was easy for these patients to develop automatic emptying of the bladder in the absence of infection of the genitourinary tract. It appeared also that in these patients no such extreme atrophy of the muscles of the lower extremities developed as was observed, for instance, in patients with anterior rhizotomies of the cauda equina.

I examined a patient in 1947 who in 1931 had suffered a fracture dislocation of the eleventh dorsal vertebra, with physiologic severance of the spinal cord between the tenth and eleventh dorsal segments. Spastic paraplegia had developed, and there existed a terrific constant pain referred to the anesthetic and paralytic areas. This pain would be made worse by any movement that might increase the tension on the spinal cord. The site of the lesion had never been explored, so a laminectomy was done for this purpose. At the levels of the eleventh and twelfth dorsal vertebrae there existed a small fibrotic cord surrounded by dense fibrous arachnoiditis, but no spinal fluid. Further exploration revealed the presence of spinal fluid near the upper border of the tenth dorsal vertebra.

I was confronted by the problem of doing a procedure that would eliminate pain and the spastic paraplegia. An anterior rhizotomy would require additional exploration and would not relieve the pain. A chordotomy would require another operation. I recalled my former experience with the coal miners and resected that portion of the fibrotic spinal cord that lay behind the body of the twelfth dorsal vertebra. I attempted to make a new cul-de-

sac for the proximal end of the severed cord, in the hope that the presence of spinal fluid would prevent the recurrence of adhesive arachnoditis at that level.

The patient was completely relieved of the pain and the spastic paraplegia. The automatic emptying of the bladder continued unchanged. Subsequently he was able to enjoy a limited social life.

During 1950 he began to have sharp pains in the groins, due presumably to the recurrence of adhesive arachnoiditis about the fibrotic proximal end of the severed spinal cord. In April 1953 I performed a bilateral chordotomy to relieve the pain. At that time I attempted to cut the sympathetic tracts in the spinal cord to produce a better blood flow to the lower extremities.

(The aforementioned coal miners with flaccid paraplegia would frequently exhibit such severe vasoconstriction of the lower extremities, with resulting nutritional disturbances, requiring sympathectomy by injections of alcohol into the appropriate lumbar paravertebral ganglia.)

This patient is at the Home for Incurables in Washington, where he has been since 1931 and where he enjoys a fairly active social life. The lower extremities are still flaccid, and extreme atrophy of the muscles has not occurred.

I congratulate Dr. Cooper on his excellent paper. I wish that he would give us his indications for subtotal cordectomy. I have recommended the type of subtotal cordectomy just described for patients who have had complete physiologic transection of the cord at a higher level with resulting spastic paraplegia. This type of operation is never recommended for a patient who has any sensation or any voluntary movement in the lower extremities.

CLOSING REMARKS BY DR. COOPER

DR. COOPER: I don't know whether I mentioned one of the patients in this group who was operated on by Dr. Hoen, who performed subtotal cordectomy. A completely satisfactory automatic bladder did develop, although a *per diem* maneuver was necessary. The only indication I can give for the procedure is what I mentioned. I think that subarachnoid alcohol injections should be employed. If there is a recurrence and one has to operate, I think, just as a matter of expediency, that it is easier and more nearly foolproof to do this than to do an anterior rhizotomy. It can be accomplished more quickly and requires less effort.

RÉSUMÉ

L'auteur passe en revue les causes physiologiques, les manifestations cliniques et le traitement neuro-chirurgical de la spasticité dans les paraplégies. L'intervention neuro-chirurgicale est indiquée lorsque d'une part les muscles ne se contractent plus volontairement et d'autre part lorsque, par la physiothérapie, le traitement conservateur échoue. Les principales méthodes neuro-chirurgicales pour lutter contre la spasticité sont: la neurectomie périphérique, les injections d'alcool sousarachnoïdiennes, la rhizotomie sélective antérieure et la cordotomie subtotale. Toutes ces méthodes appliquées judicieusement apporteront un soulagement de la spasticité et une amélioration dans le traitement du paraplégique.

ZUSAMMENFASSUNG

Es wird eine Übersicht über die physiologischen Grundlagen, die klinischen Erscheinungen und die nervenchirurgische Behandlung der Muskelspasmen bei Lähmungen der unteren Extremitäten gegeben. Die spastische Muskellähmung erfordert chirurgisches Eingreifen, wenn eine befriedigende Spontanheilung sich als unmöglich herausstellt, wenn konservative Behandlung erfolglos ist, und wenn die befallenen Muskeln keine nützliche kontrollierte willkürliche Funktion aufweisen. Die wichtigsten nervenchirurgischen Methoden zur Behebung der Spasmen sind die periphere Nervenresektion, die subarachnoide Alkoholeinspritzung, die selektive Durchschneidung der Vorderwurzeln und die subtotale Rückenmarksresektion. Bei wohlüberlegter Auswahl und Anwendung des geeigneten Verfahrens kann ausnahmslos eine Linderung der Spasmen und eine bessere Versorgung des gelähmten Kranken erwartet werden.

RESUMO

São revistos pelo autôr neste trabalho, a fisiologia, as manifestações clínicas e o tratamento neuro-cirurgico da espasticidade nos casos de paraplegia.

Estandartisa a indicação do tratamento neuro-cirúrgico em tais casos, citando como técnicas mais preconisadas a neurectomia periferica, a injeção subaracnoidea de alcool, rizotomia anterior seletiva, e a cordectomia espinhal subtotal.

Refere-se por fim aos resultados obtidos com tais metodos cirúrgicos.

RESUMEN

Se revisa el fondo fisiológico, manifestaciones clínicas y tratamiento neuroquirúrgico de la espasticidad en la paraplejia. La espasticidad en paraplejia requiere tratamiento quirúrgico cuando hace imposible una rehabilitación adecuada, no responde al tratamiento conservador y complica músculos que no tienen una función voluntaria útil. Los principales métodos neuroquirúrgicos para el alivio de la espasticidad son, la neurectomía periférica, la inyección subaracnoidea de alcohol, la rizotomía anterior selectiva y la cordectomía medular subtotal. El método apropiado cuidadosamente seleccionado y aplicado, producirá invariablemente un alivio de espasticidad y mejoramiento del cuidado del paciente parapléjico.

RIASSUNTO

E' stata trattata la fisiopatologia, la clinica e la cura neurochiurgica delle manifestazioni spastiche nella paraplegia. Tali lesioni richiedono l'intervento quando sono tali da rendere impossibile la rieducazione del paziente, quando resistono alla terapia conservativa e quando interessano muscoli a funzione volontaria non controllata. I metodi usati per vincere gli spasmi sono i seguenti: neurectomia periferica, iniezioni subaracnoidee di alcool, rizotomie anteriori elettive, cordotomie subtotali. Questi metodi, impiegati selettivamente, portano invariabilmente al miglioramento degli spasmi.

Common-sense nerve fibers are seldom medullated before forty—they are never seen even with a microscope before twenty.

Dermatographia is an outward and visible sign of an inward and hidden instability. We expect it in medical students about the month of May just before examinations.

—*Osler*

Surgical Treatment of Congenital Spinal Disorders*

DONALD D. MATSON, M.D., F.A.C.S.

BOSTON, MASSACHUSETTS

BECAUSE the nervous system continues to grow and mature after birth, it is important to recognize any congenital disorders that may give rise to subsequent complications or progressive neurologic dysfunction as growth proceeds and to carry out corrective surgical procedures early in life whenever possible. Certain types of spina bifida occulta are accompanied by intraspinal lipomas, intraspinal meningoceles, fibrous stalks, dermoid cysts, bony spicules (diastematomyelia), or other lesions which, by compression or distortion of the growing elements of the neural axis, fall into this category. Early investigation and conservative surgical treatment are to be stressed.

Persistence of a congenital dermal sinus tract from the midline of the skin overlying the spine into the meninges or into the spinal cord itself may lead to infection within the central nervous system or the meninges. In addition, there may be expansion of the inner terminus of such a sinus into a cyst, which may act as a mass lesion in addition to being a focus for abscess formation. Prophylactic removal of all persistent dermal sinus tracts leading into the spinal canal is strongly recommended.

There is no routine treatment for meningoceles and myelomeningoceles associated with spina bifida; each constitutes a problem of its own, and the method and time for surgical treatment depend on many factors, such as the size and level of the lesion, the type of covering, the condition of the surrounding skin, the amount of neural involvement, the adequacy of the cerebrospinal fluid circulation (hydrocephalus), the economic and social status of the family, and so on. Rarely, meningoceles occur anteriorly as well as posteriorly, and this should be kept in mind in the consideration of undiagnosed masses in the mediastinum and in the retroperitoneal and retropelvic tissues.

Congenital tumors of the spinal axis warrant special attention, because there is a great temptation for the surgeon to be too radical in attempting to remove these benign lesions and thus make his patient worse. Dermoid and teratomatous lesions of the spinal canal should be approached conservatively, with emphasis on the contents of cysts, removal of the solid tumor and cyst wall which is freely mobilized, with no attempt at sharp dissection from neural tissue itself. This type of conservative management may be followed by an excellent return of neural function persisting for many years.

DISCUSSION BY DR. H. E. LEFEVER, COLUMBUS, OHIO

DR. LEFEVER: I should like to limit my discussion of Dr. Matson's paper on stressing the relation between spina bifida occulta or aperta and intraspinal lipomas. My interest in this subject was quickened as I reviewed my series of 228 cases of intraspinal neoplasms and to my surprise found that 7 were lipomas. Three lipomas were found underlying spina bifida occulta; 3 accompanied spina bifida aperta, and only 1 was unrelated to a dysraphic defect. One of the 7 was of the rare intradural type, of which 30-odd have been reported. Two intraspinal lipomas coexisted with midline subcutaneous lipomas in the same metameres as the intra-

*Abstract of paper read at the Eighteenth Annual Congress of the United States and Canadian Sections, International College of Surgeons, New Nork, Sept. 13-17, 1953.
Submitted for publication April 24, 1954.

spinal mass. In 3 hirsutism was also present, overlying the defect.

The clinical manifestations of intraspinal lipomas may differ from those of other intraspinal mass lesions in that root pains are uncommon despite the frequent involvement of the roots by the tumor and despite the fact that the intravertebral foramen is frequently filled with tumor tissue. Complete subarachnoid block is unusual, despite the large size of the tumor mass. The spinal roentgenogram may reveal evidence of congenital defects, and the spinal canal may be widened for a considerable distance in the region of the lipoma.

Histologically the tumors are composed of large, polygonal, closely approximated, translucent elements, seen in normal adult fat. In Dr. Elsberg's series of 254 intraspinal neoplasms, 4 were lipomas and 1 was related to spina bifida. In a series of 557 intraspinal tumors reported from the Mayo Clinic by Rasmussen, Kernahan and Adson, 4 lipomas were encountered. Three of these were related to congenital defects. It is the consensus that a congenital factor is present in the inception of most intraspinal lipomas, as is evidenced by the common association with spina bifida, cleft palate, harelip and multiple subcutaneous lipomas. Intraspinal lipomas constitute approximately 1 per cent of intraspinal neoplasms.

Should a roentgenographic survey of the spinal canal reveal spina bifida occulta in a patient showing evidence of an intraspinal compression syndrome, the possibility of a lipoma should be seriously considered.

CLOSING REMARKS BY DR. MATSON

DR. MATSON: I like to think that the way to approach lesions associated with spina bifida occulta is radical investigation combined with very conservative surgical treatment. There certainly is little or nothing to be gained by manipulation of the cord itself or the cauda equina, whether the lesion is a dermoid cyst or one of the so-called lipomas (I like to avoid this term because I think we may get to think of them as discrete tumors, which they are not). These fatty masses may extend everywhere from the skin right on down into the substance of the cauda equina, and one may injure the neural tissue any time after the skin incision is made if he is not aware of the simple fact that the overlying fascia, muscle, bone and meninges may all be defective.

In so far as one can remove any structures that are compressing or distorting the cord or the cauda equina, divide fibrous bands, remove intraspinal meningoceles, excise discrete masses of fat, and remove dermoid cysts, surgical intervention is extremely worth while, but even then it must be carried out very carefully. Beyond that, I think it is meddlesome to feel that one must explore every patient simply because neurologic signs are present.

If Dr. Williams would like his problem of enuresis reduced to statistical terms, I certainly can't do it at this time. In a growing child there are all sorts of possibilities, but I think there isn't any question but that persistent enuresis, particularly if it has come on after a period of satisfactory bladder training and there are normal examinations of the urine and urinary tract, demands consideration of the possibility of a neurologic lesion. Enuresis alone, occurrying without any evidence of other bladder disturbance or musculoskeletal disturbance, I should think is pretty rare as a result of spina bifida, congenital tumor or other lesion involving the cauda equina.

Thought is the labor of the intellect, reverie is its pleasure.

—*Hugo*

The soul is dyed with the color of its leisure thoughts.

—*Inge*

Bladder Disturbances and the Management of Patients With Injury to the Spinal Cord *

ERNST BORS, M.D.

LONG BEACH, CALIFORNIA

IN summarizing my current policy as to the treatment of the traumatic cord bladder, I arrived at the following outline: Routine treatment should be instituted for upper and lower motor neuron lesions alike, preferably with tidal drainage. Permanent suprapubic cystostomy has been relegated to cases of cervical injuries that fail to respond to all efforts toward restoring bladder function or to cases in which there is incorrigible obstructive disease of the lower portion of the tract. Routine application of suprapubic cystostomy, still practiced by some, does not necessarily destroy the prospect of a balanced bladder function, but it postpones the achievement of this goal.

If routine treatment fails in a patient with a lower motor neuron lesion, transurethral resection is the procedure of choice.

If routine treatment fails in a patient with an upper motor neuron lesion, I employ the following procedures, in order of preference:

(1) Pudendal nerve anesthesia; if no permanent result occurs, (2) transurethral resection, with special consideration of the anterior lip; if this fails, (3) repetition of pudendal anesthesia with subsequent section of the pudendal nerve on the dominant side of the body (provided that the result from anesthesia was only temporary), and finally (4) section of the remaining pudendal nerve when all preceding interventions have failed.

*Abstract of paper read at the Eighteenth Annual Congress of the United States and Canadian Sections, International College of Surgeons, New Nork, Sept. 13-17, 1953 [J. Internat. Coll. Surgeons *21*:513-527 (April) 1954].

DISCUSSION BY DR. THOMAS I. HOEN, NEW YORK

DR. HOEN: It is a privilege to appear on this program in connection with Dr. Bors' presentation. My colleagues and I have been interested in paraplegia since the beginning of the war, when we organized the paraplegic center at the U. S. Naval Hospital at St. Albans. Our interest has continued, but I realize, after hearing Dr. Bors present 844 cases in which he has personally treated these conditions, that we are comparative amateurs.

I find it difficult to discuss this paper, with all the data that have been presented, and shall limit myself to a few comments. Dr. Bors has used all the known methods of treating the neurogenic bladder, and, although he has not himself employed all of them, the ones he has used are the commonly accepted ones, and I think that it is valuable to know the results.

I am surprised to know that Dr. Bors does as many transurethral resections as he does. I know that during the war Thompson resorted to that procedure in an excessive number of cases, and considering it the answer to all problems of bladder management. My associates and I took rather an opposite stand, I think with justification. We studied our cases very carefully with cystourethrograms, and this enabled us to establish criteria and precise indications for resection of the vesical neck. Some of the patients with retention did not have the hypertrophied internal sphincter described by Thompson. Quite to the contrary, they had what we called a "funnel-neck bladder," with the internal sphincter wide open around the brim of the funnel. Obviously, resection of that internal sphincter could accomplish nothing toward relief of retention. We were of the opinion that in those cases the retention was due to spasticity of the external sphincter (the striated muscle), and for that reason we devoted our attention to relief of the spasm of this muscle by interruption of the lower motor neuron supply, with varying results. Our procedure

was to block the sacral nerve roots separately, with a view to relaxation of the sphincter. A clear-cut result, with ability to empty the bladder, was considered an indication for intradural anterior rhizotomy. In this case it was evident that muscle spasm was present without fibrosis. However, our results were not consistent and left us somewhat confused in our thinking.

I could wish that Dr. Bors had classified his cases slightly differently from a neurologic point of view. I think it is a little unfortunate that he talked about cervical, upper thoracic, lower thoracic and lumbosacral lesions. This fails to recognize an area of importance and a definite type of injury which may be called the conus lesion. A lesion at twelfth dorsal level is apt to be a conus lesion. A lesion at the first lumbar is apt to be a conus lesion. To separate the twelfth dorsal from the first lumbar is apt to confuse the picture. I should prefer to classify these cases as: (1) cervical lesions, in which the entire sympathetic outflow is below the lesion; (2) lower thoracic lesion, in which a good bit of the sympathetic supply is above the lesion; (3) the conus lesion, in which there is destruction of the anterior horn cells involved in bladder function, and (4) the cauda equina lesion, a true peripheral nerve lesion as far as the motor supply is concerned (it is not a peripheral nerve as far as the sensory supply is concerned, because the injury is central to the sensory root ganglion). I think that if Dr. Bors' cases were classified in this way I should find it easier to follow some of the rationale of his treatment.

Unquestionably, it is most fortunate that a urologist of Dr. Bors' standing has devoted himself to this neurologic problem, and I trust that his association with neurologists and neurosurgeons has been as pleasant as it has been productive.

DR. HAROLD C. VORIS, CHICAGO: I should like to add my personal thanks to Dr. Bors for having come to us and for presenting, as a urologist, such an excellent paper on this problem, which certainly can be solved only by the continued cooperation of both urologists and neurosurgeons.

CLOSING REMARKS BY DR. BORS

DR. BORS: Dr. Hoen is quite right about the transurethral resection. I, too, am against its indiscriminate use. At the time when Thompson performed it in about 50 per cent of his cases, I did resections is only 5 per cent of mine. But as time goes on, changes take place that make resection necessary. I didn't mention that resection may become necessary at any time from seven months up to three hundred and sixty months after injury, and that is a long interval. After that interval resection becomes necessary not because of the striated muscles, which can be treated as I indicated in my presentation, but because of the smooth muscles, which become secondarily contracted, especially in those cases in which an upper motor neuron lesion of the conus has been converted into a lower motor neuron lesion. That still leaves the smooth muscles of the vesical neck entirely supplied by the sympathetic nerves, and consequently those muscles eventually become hypertrophied. Perhaps I did not make myself clear on that point.

As far as the conus is concerned, I fully concur with you. However, the term "upper or lower motor neuron lesions" refers to the conus. The additional differentiation of cervical, upper or lower thoracic and lumbosacral lesions still has its merits, because it describes the original level of the lesion; this in turn may be associated with an upper or a lower motor neuron lesion of the conus. At present I know of no clinical method, so far as bladder function is concerned, to distinguish a lesion of the conus from one of the cauda equina.

It is better to give than to lend, and it costs about the same.

—Sir Philip Gibbs

When some men discharge an obligation you can hear the report for miles around.

—Mark Twain

Evaluation of Skeletal Traction in the Treatment of Cervical Spine Injuries*

CHARLOTTESVILLE, VIRGINIA

WILLIAM GAYLE CRUTCHFIELD, M.D.

WHEN the spinal cord is severely injured at the level of the atlas or axis, it is probable that the patient dies immediately or at least before a clinical diagnosis can be made. Therefore, patients with compression fractures or dislocations high in the cervical spine show little, if any, evidence of spinal cord injury. Having the responsibility of a patient who has barely escaped a tragic end and whose injury is such that improper or inadequate treatment could result in sudden death, the attending surgeon is faced with a serious problem. I have no quarrel with those who believe that these patients should be treated by means of spinal fusion. In fact, my associate, aided by members of the department of orthopedic surgery, has operated on five of our patients. Good results were obtained, but on the other hand equally satisfactory results were obtained in a much larger series of cases treated conservatively. Moreover, throughout my experience with injuries of this type, which includes a period when treatment consisted only of applying a plaster support, no untoward effects from conservative treatment have been observed. Now that better methods of handling these patients are available, there seems to be less reason than ever for resorting to spinal fusion operations, which are by no means free of risk. The patient who has a satisfactory fusion operation may be able to discard his brace a little sooner than the one treated conservatively, but this is of minor importance when compared with the disadvantages of the major surgical procedure.

Compression fractures of the atlas and axis are more responsive to conservative treatment than are dislocations at these levels. They tend to remain in a satisfactory position and hence require a shorter period of traction, whereas dislocations are more apt to be accompanied by greater injury to the spinal ligaments that permits the vertebrae to slip out of alignment unless traction is maintained for a longer period. Injuries at this high level require very little traction, and the patients are comfortable except for their enforced bed rest. It has been our policy to continue traction for 8 to 12 weeks depending on the type and severity of the injury and then to rely on a light adjustable brace. Some patients are allowed to discard the brace after three months. Others are required to use it for six months or longer, depending on individual circumstances.

SUMMARY

When properly applied, skeletal traction is the safest, most effective, and simplest method of treating patients with acute injuries of the cervical spine. Furthermore, when used to its full advantage it leaves few, if any, indications for laminectomy, spinal fusion, or plaster supports.

DISCUSSSION BY DR. RALPH B. CLOWARD, HONOLULU

DR. CLOWARD: Many of Dr. Crutchfield's comments and suggestions are indeed welcome. In my particular locale, where we swim in the ocean the year round, injuries to the cervical portion of the spine are frequent. I probably encounter as many injuries of this

*Reprinted by permission of the Journal of the American Medical Association.

type as do the majority of neurosurgeons, or more. I am perplexed at times to know just how to handle some of them. The first puzzle Dr. Crutchfield has answered for me is why these beautiful little tongs, which he devised are continually slipping out of the skull. I fell heir to a half dozen pairs of his tongs right after the beginning of the war. Apparently they were the second edition, so to speak, of the Crutchfield tongs. Having too much respect for Dr. Crutchfield and his invention to complain, I never bothered to write him to ask why they kept coming out, so I proceeded to alter them so that they would not. I threw away the small drill which is used to make the hole in the skull. Through a 1 cm. scalp incision a trephine hole was made in the skull with a small Hudson drill, cutting through the outer table and the diploe down to the inner table. I copied the Blackshire tongs and had a blunt end made to replace the sharp end the Crutchfield tongs. The blunt end does not wander on into the skull or slip out after it is engaged in the larger hole.

Another point I feel we should heed in Dr. Crutchfield's paper is the amount of weight used to reduce the dislocation. Certainly, leaving 15 to 20 pounds (6.8 to 9.1 Kg.) of weight on the patient for three, four or five weeks does interfere with proper healing. I was not smart enough, I must humbly admit, to realize why the fracture would not heal after the patient had been in traction for six weeks. It was distressing to find the dislocation drifting back to where it was in the beginning after traction had been removed.

One patient, a man 27 years old, had a rather severe fractured dislocation between the fifth and sixth cervical levels. No cord damage could be demonstrated, but severe pain and paresthesias were present in both upper extremities. The fracture was reduced and the alignment of the spine returned to normal with about 15 to 18 pounds (6.8 to 8.2 Kg.) of weight. Complete cessation of complaints and subjective symptoms was immediately obtained. The patient remained in traction for about six weeks. After the traction had been applied, there was marked increase in the width of the intervertebral space at the level of the fracture. As Dr. Crutchfield has pointed out, this probably is indicative of a tear of the anterior ligaments.

The traction was discontinued after six weeks; a Thomas collar was applied, and the patient was permitted to get out of bed. Within forty-eight hours after he first sat up, the pain, paresthesias and weakness in the upper extremities returned. A roentgenogram was obtained which demonstrated the body of the fifth cervical vertebra slipping forward on the sixth; the condition was as bad as it had been, six weeks before, when the patient was admitted to the hospital, or worse. There was a very wide space between the spinous processes of the adjacent vertebra at the level of the fracture.

The patient was returned to bed, the Crutchfield tongs reapplied and 15 pounds (6.8 Kg.) of traction used. The roentgenogram again showed the spine nicely aligned, and the same satisfactory clinical result was obtained—that is, complete disappearance of symptoms. The patient was kept in traction for another six weeks. This made a total of three months in bed. The next time he was permitted to get up the same thing happened. The pain and paresthesias in the arm and the weakness of the grip returned, there was recurrence of the dislocation, with obvious evidence that the fracture had failed to heal. The patient was then subjected to an open surgical operation. The dislocation was corrected by pulling the spinous processes of the fifth and sixth cervical vertebrae together and securing them firmly with a heavy stainless steel wire. Posterior spinal fusion was effected by placing bone over the adjacent laminae and spinous processes.

In all, this patient was hospitalized for approximately five and a half months before his fractured dislocation was healed. Because of my experience with this patient it became my practice to treat these lower cervical injuries—that is, the severe fractured dislocations of the bodies with little cord involvement but with definite evidence of root pressure—by early operation—wiring the spinous processes and fusing the laminae, with a greater chance of recovery and a shorter period of hospitalization. The patient is kept in skull traction while the spine is healing. In this way he is required to remain in bed only a month or six weeks.

The patient whom I have just described was operated upon in 1944. All conditions similar to his have been treated in this manner since that time. As a result of Dr. Crutchfield's remarks I may be able to save myself considerable work and my patients unnecessary surgical treatment by reducing the traction weight from 15 to 5 pounds (6.8 to 2.3 Kg.) after the first two days, if this will permit the torn ligaments to heal in a short time.

The practice of taking immediate roentgenograms to determine whether or not the fracture is reduced is a good one. If the interspace is too wide at the level of the fracture, the weight may be reduced. In Dr. Crutchfield's opinion, if a small amount of weight is kept on the patient for six weeks, the ligaments may heal and hold the fracture, thus making an open operation unnecessary.

Of course, there are always two schools of thought on every subject—whether one should operate on these patients early, wire the fragaments together, fuse the spine and *then* let them heal, or whether traction should be applied and the fracture be permitted to heal without operation. It would seem that the decision depends entirely on the individual case and the individual approach of the surgeon.

DISCUSSION BY DR. THOMAS I. HOEN, NEW YORK

DR. HOEN: It has been very interesting to hear first Dr. Crutchfield and then Dr. Cloward discuss skeletal traction. Before Crutchfield tongs were available, I applied traction by putting wires through burr holes in the skull. I think this method still preferable if the patient is to be kept in traction for six weeks or more as it is less irritating to the soft tissues and eliminates the possibility of slipping or brain injury. There is also a lower incidence of infection. However, I do use the tongs and know that they are the most valuable apparatus for quick application of skeletal traction.

With regard to management of the patient after application of traction, my policy has been to reduce the weight quickly after reduction has been accomplished, and to follow the patient with roentgenograms every six hours to see whether the position is held. If the position can be maintained with light traction for a time, I agree as Dr. Crutchfield, but if there is any tendency for the dislocation to recur with the reduced weight, I do then what Dr. Cloward does routinely—resort to open exposure and fixation of the spinous processes. Not all of these cases are the same. It depends a great deal on the fracture associated with the dislocation. If the facets or pedicles are fractured and do not give any support, the dislocation is apt to recur; but when the structures are minimally damaged good alignment after reduction is easy to maintain without the necessity of operation for internal fixation.

It might be interesting just to mention that we might all have begun using skeletal traction before we did, if it had not been for Dr. Cushing! In 1930, after a Harvard graduation celebration, one of the boys dove into the family swimming pool, in which there was no water. He arrived at the Peter Bent Brigham Hospital shortly afterward, accompanied by many relatives and friends and a number of prominent Boston physicians. Dr. Cushing was in a great dither, and all of us, including Dr. Horrax, were milling about trying to reduce a very bad fracture dislocation, without success. During a lull in the activities I said in a low voice to Dr. Cushing, "Why don't we get a pair of ice tongs like they hang the bodies up with in the morgue, fix them in his skull, and stretch him out a little bit?" He looked at me and said, "Hoen, what a horrible thought." Needless to say, several years elapsed before I thought of it again.

Many people enjoy nothing of their happiness, but the fear of losing it.

—*Rivarol*

Human felicity is produc'd not so much by great pieces of good fortune that seldom happen, as by little advantages that occur every day.

—*Franklin*

The secret of being miserable is to have leisure to bother about whether you are happy or not.

—*Shaw*

Experimental Regeneration in the Spinal Cord*

W. W. CHAMBERS, Ph.D.

PHILADELPHIA, PENNSYLVANIA

EXPERIMENTS on central neuron regeneration, utilizing the bacterial polysaccharide Pyromen, have shown that this substance causes proliferation of undifferentiated connective tissue and phagocytes, and apparently by this mechanism prevents the formation of glial scar at the site of central injury.

The secondary effect of such glial inhibition is the regeneration, in limited quantity, of central neurons. The factors responsible for the recovery of these neurons after injury and their subsequent capacity for regrowth are not at present known, but do not appear to be due to the direct action of Pyromen. The capacity of central neurons for regeneration is strikingly poor, even when, under the influence of Pyromen, an apparently adequate substratum is present for the growth of their processes.

The main problem in regeneration of the central nervous system in adult mammals lies in the apparently limited capacity of the neuron itself to survive injury, and subsequently to show vigorous and active growth. An integral part of the problem is the intimate relation between neurons and glia. A most promising approach to the solution of this important problem is a careful study of the marked regenerative capacity in amphibia.

DISCUSSION BY DR. O. H. FULCHER, WASHINGTON, D. C.

DR. FULCHER: Until June 1952, when I heard Dr. Windle's presentation at Atlantic City, I thought that any idea of regeneration of the spinal cord was obsolete. That this question should be revived is electrifying to anyone who has cared for paraplegic patients. I returned from Atlantic City feeling new hope, even though the success of any application of the method to man might be remote indeed. Any hope for aid to the paraplegic patient must be kept alive.

It was with this feeling of responsibility that I decided to use Pyromen clinically. My only knowledge of the drug and of the therapeutic rationale had been acquired at this one meeting. I have now treated 16 patients, but only 7 will qualify for pertinent discussion.

1. P. O., the 15-year-old son of a physician, was injured in an automobile accident on July 31, 1950. He suffered a fracture-dislocation of the twelfth thoracic vertebra with physiologic transection of the spinal cord between the eleventh and twelfth dorsal segments. A laminectomy was performed on July 31 and again on August 18. The second exploration revealed necrosis of the cord at the level of the fracture-dislocation. Pyromen was begun on Aug. 13, 1952. It was evident that the drug could do little at this late date if its sole therapeutic action consisted of preventing the formation of glial tissue. This patient tolerated large doses of the drug over a period of many months, without any subjective or demonstrable benefit.

2. F. S., a man aged 64, had partial upper and lower motor neuron paralysis resulting from Paget's disease of the dorsolumbar portion of the spine and a ruptured third lumbar disc. A laminectomy was performed on Dec. 1, 1951, without resulting improvement. Pyromen was started on Oct. 2, 1952, and continued for four months, without demonstrable improvement.

3. W. M., a man 65 years of age, underwent operation on March 15, 1951, for rupture of the sixth cervical disc, associated with severe hypertrophic arthritis. There was evidence of injury to the spinal cord as well as severe radiculitis. He was given only three injections of Pyromen, because of severe reactions. For some reason, however, this patient subsequently had much less pain.

4. J. S., a youth 18 years old, suffered a fracture-dislocation of the second lumbar vertebra when a pile of plywood overturned on

*Abstract of paper read at the Eighteenth Annual Congress of the United States and Canadian Sections, International College of Surgeons, New Nork, Sept. 13-17, 1953 [J. Internat. Coll. Surgeons *21*:225-229 (Feb.) 1954].

him. Paralysis and anesthesia were present on the right, below the level of the injury. A laminectomy was performed immediately to decompress the cauda equina and the conus of the cord. Pyromen was begun immediately. A remarkable recovery has occurred. There is no proof that the drug was effective, as young patients sometimes improve phenomenally after injuries to the nervous system.

5. H. D., a man 34 years of age, had persistent "saddle-shaped anesthesia extending to the second sacral segment bilaterally. This condition followed spinal anesthesia for removal of a ruptured intervertebral disc at the fifth lumbar interspace. The patient was highly allergic, which at first made him reluctant to take any drugs. There had been practically no improvement until Sept. 8, 1952, when Pyromen was first employed. He has continued with this drug up to the present time, with phenomenal improvement. It is possible, of course, that the use of Pyromen was just coincident with spontaneous improvement, but this does not appear likely, either to me or to the patient.

6. B. K., a girl 14 years old, on June 6, 1952, suffered a compressed fracture of the first lumbar vertebra, with severe dislocation, when she was "jackknifed" by the falling tailgate of a truck. At first there was complete physiologic transection of the conus. A laminectomy was performed to decompress the cord. The use of Pyromen was begun on October 2. This patient has had a rather remarkable recovery. It is possible that the improvement would have developed anyway, considering the youth of the patient.

7. T. D., a 61-year-old man, was injured on June 8, 1948. He suffered a compressed fracture of the body of the fifth thoracic vertebra with complete physiologic severance of the cord at that level. A laminectomy was performed a few hours after the injury to decompress the cauda equina. Subsequently infectious hepatitis developed; also bed sores, which required plastic surgical treatment. On Dec. 15, 1950, a myelogram was taken because this patient had ceased to improve and because he was beginning to suffer with massive reflex motions of the lower extremities. The myelogram revealed partial block at the site of the old injury. On Dec. 11, 1951, the cord was explored at the site of the partial block. It was somewhat smaller than normal and was adherent to the meninges on the right. The patient made some improvement after this operation, but the spastic paraplegia became worse and the reflex mass movements of the lower extremities increased. Pyromen was begun on Aug. 13, 1952, and has been continued ever since. There has been great improvement. The spastic paraplegia has largely disappeared, and the reflex massive movements are no longer troublesome. He has regained sensation to touch over the lower part of the trunk, the entire right leg, and the left leg down to the knee. It is exceedingly difficult to explain the improvement in this patient, which has occurred four years after the injury, if the only function of the drug is the prevention of the formation of glial tissue. I am fairly well convinced that Pyromen did cause the improvement. It would appear, therefore, that this drug must have some other action of value.

Many of our cares are but a morbid way of looking at our privileges.
—*Scott*

To carry care to bed is to sleep with a pack on your back.
—*Haliburton*

Worry is interest paid on trouble before it falls due.
—*Inge*

I have never yet met a healthy person who worried very much about his health, or a really good person who worried much about his own soul.
—*Haldane*

Tumors of the Brain and Spinal Cord as They Concern the General Surgeon

HENDRIK J. SVIEN, M.D., F.A.C.S.*
AND ANDREW L. KARAVITIS, M.D.**
ROCHESTER, MINNESOTA

IN this day of specialization in medicine, the trend is for each specialist to concentrate increasingly on the particular field he has chosen. This trend, of course, has been beneficial in many ways. Nonetheless, it is prudent now and then to survey one's course in any specialty, and to reflect that diseases and pathologic processes still take no heed of specialization. That is, they still manifest themselves in symptoms that are quite nonspecific.

Let us consider some examples. To the general surgeon, the symptoms of nausea and vomiting suggest appendicitis, disease of the gallbladder or obstruction of the bowel. To the neurosurgeon, however, the same symptoms suggest increased intracranial pressure. Pain in the right upper abdominal quadrant causes the general surgeon to think of a diseased gallbladder, but the same pain makes the neurosurgeon speculate on the presence of a tumor of the spinal cord. One could go on similarly with other symptoms — headache, pain in the precordial region and the like.

Fortunately, in most instances of appendicitis or disease of the gallbladder, other symptoms and signs besides the nausea and vomiting are present to make the true diagnosis quite certain. Yet there are cases in which the situation is called "borderline" — meaning that the associated signs and symptoms are not fully convincing and the diagnosis is not clear. It is in this borderline type of situation that physicians in different specialties must work together for solution of the problem in the best interests of the patient. A few cases which present so-called borderline situations will show what is meant and the problems involved.

Man is not perfect; if he were, physicians and surgeons would have nothing to do. Still less do we expect surgeons to be perfect in their mastery of every possible surgical situation that may arise. It is known and recognized, in fact, that no one man today is able to master every conceivable ramification of surgery; were it otherwise, there would be no surgical specialties. The general surgeon performs with ease a procedure that might be strange to the neurosurgeon; conversely, the neurosurgeon has amassed certain skills and a body of special knowledge that might never be demanded of the general surgeon.

For these reasons, the illustrative cases to be presented here reflect discredit on no one. Quite to the contrary, they only point up again what has been known for many years: that when a particularly stubborn diagnostic problem challenges the individual practitioner in any field, the virtues of consultation can be very great.

REPORT OF CASES

CASE 1.—Recently we saw in consultation a patient who had undergone cholecystectomy four weeks before. He was a recent immigrant, a fact which only complicated the difficulties of diagnosis by interposing a language barrier. This man had some epigastric distress after heavy meals for several years, but had not had pain in the right upper abdominal quadrant or episodes of jaundice. Three weeks

From the Section of Neurologic Surgery, the Mayo Clinic and the Mayo Foundation. (The Mayo Foundation is a part of the Graduate School of the University of Minnesota.
*Fellow in Neurosurgery.
Read at a regional meeting of the International College of Surgeons, Oklahoma City, Oklahoma, January 1954.
Submitted for publication April 24, 1954.

before the cholecystectomy nausea, vomiting and some headache had developed. The nausea and vomiting occurred daily. Cholecystograms had been taken, and they revealed gallstones. Cholecystectomy was carried out. Yet nausea and vomiting persisted, as did the headache.

Headache can be indicative of much or of nothing; many persons have headaches; many, for instance, who have indigestion complain of headaches. However, the headache of this man was typical of the headache of brain tumor. It was present in the frontal and temporal areas and was worse on the right side. It came on daily, in the early morning, and frequently awakened the patient at 4 a.m.

The patient also said that he had periods of double vision at this time. Double vision also is a symptom of brain tumor, which produces increased intracranial pressure. This particular defect in vision is due to pressure on the sixth intracranial nerve, which innervates the lateral rectus muscle of the eyeball. This nerve has a long intracranial course and is often the first to be affected by increased intracranial pressure.

Neurologic examination disclosed minimal left hemiparesis. Funduscopic examination revealed chronically choked ocular discs of 3 diopters. It is highly probable that some degree of edema of the optic discs had been present at the time of cholecystectomy. Roentgenograms of the skull revealed a shift of the calcified pineal gland to the left, indicating displacement by a mass on the right (Fig. 1). Electroencephalographic study revealed a delta focus in the right frontal region. A right carotid arteriogram was taken; it demonstrated displacement of the anterior cerebral artery to the left (Fig. 2). At operation a large meningioma, a benign tumor, was completely removed (Fig. 3). Because of the location of the lesion in the right frontal area, accessible for removal, the patient recovered without further neural defect. The results of neurologic examination when the patient was dismissed were essentially negative.

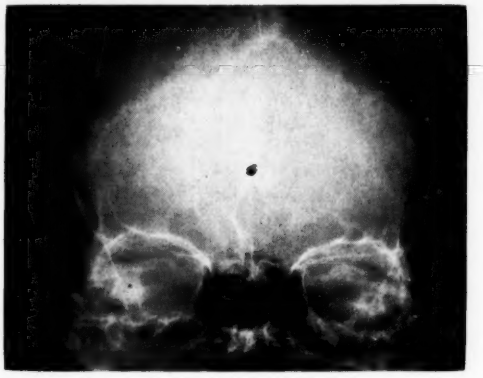


Fig. 1.—Displacement of a calcified pineal gland to the left.

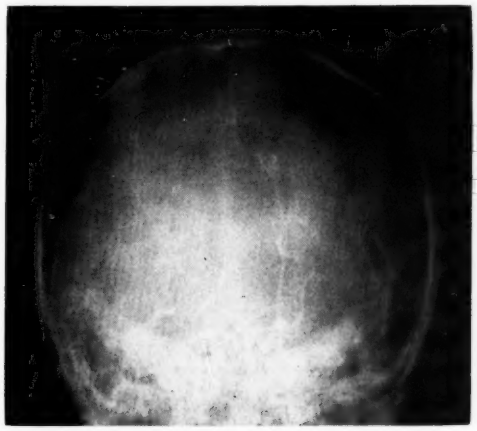


Fig. 2.—Right carotid angiogram, anteroposterior view, indicating displacement of the right anterior cerebral artery to the left, suggesting the presence of a mass in the right frontal lobe.

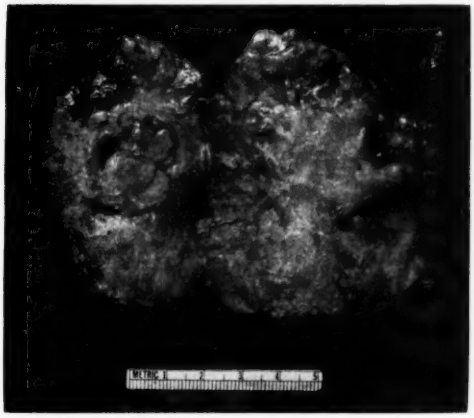


Fig. 3.—Meningioma removed in Case 1.

CASE 2.—The patient was a child, aged 2½ years, who had undergone appendectomy three weeks prior to admission because of nausea and vomiting, followed by stomachache. These symptoms had been present for about six weeks before the operation. Symptoms had not been relieved by appendectomy; on the con-

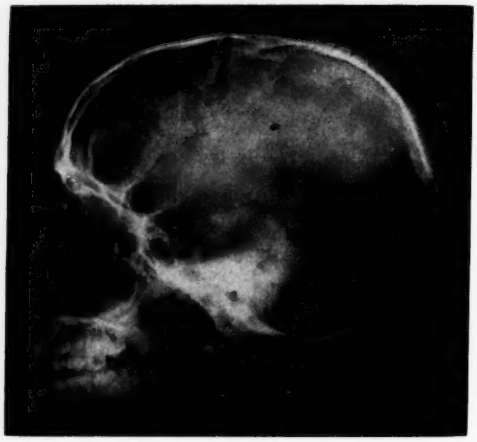


Fig. 4.—Separation of the suture lines in Case 2.

trary, they had become progressively more severe. The parents said that the headaches had been associated with the nausea and vomiting. The child would complain, in the early part of his illness, of frontal headaches and would then rather precipitously vomit. At first this chain of events would occur about once a week. During these episodes the child would be listless; he wanted to do nothing but lie perfectly still. He also was constipated during this period. The parents mentioned that in the few days before the operation the child had difficulty in feeding himself because his hands would shake and wobble when he attempted to bring food to his mouth. After discharge from the hospital, it was also noted that he had difficulty in controlling his legs. In the five days before his admission he was unable to walk at all because of this ataxia. It had been noted that during the previous six weeks he had held his neck and head unusually straight.

On examination the patient was drowsy. All the signs and symptoms necessary for a diagnosis of tumor of the posterior cerebral fossa were present—ataxia of the extremities, rigid neck, choked optic discs and separation of the suture lines of the skull (Fig. 4). In other words, the important neurologic signs were fully developed when we saw the patient, and we are sure that at the time of the appendectomy there were signs which, if not fully developed, certainly were highly suggestive of brain tumor. Had a neurologist or a neurosurgeon seen the boy at that time, the suspicion of a brain tumor as the cause of the symptoms would probably have been entertained, and attention would have been directed to the area concerned. It is highly probable that separation of the suture line and the choked optic discs were present then.

At operation a cystic astrocytoma with a mural nodule in the midline of the cerebellum was completely removed. The prognosis associated with such a lesion is hopeful, since this type of tumor is benign. However, the condition of the patient was rapidly deteriorating, and had operation been delayed for a few more days, it is doubtful that he would have survived it.

CASE 3.—A man aged 62 stated that he had lost 25 pounds (11.3 Kg.) and that anorexia had developed during the three or four months prior to his admission. Gastrointestinal roentgenograms revealed slight widening of the duodenal loop and suggested a retroperitoneal mass. Yet a mass was not detectable by palpation of the abdomen.

Abdominal surgical exploration was advised, but because the patient had complained of daily mild headaches, neurosurgical consultation was requested. The results of neurologic examination and of examination of the ocular fundi, as well as of roentgenograms of the skull, were negative, but the electroencephalogram showed left frontal dysrhythmia (Fig. 5). We decided that, although the abnormal signs were not too impressive, the presence of a brain tumor should be excluded before an abdominal operation was carried out. Ventriculograms were taken; they revealed a marked shift of the ventricular system to the right and other signs indicating the presence of a brain tumor in the left frontal area (Fig. 6). These observations—the widening of the duodenal loop and the evidence of an intracranial mass— could be well explained by a metastatic process from a retroperitoneal carcinoma. Craniotomy, however, was done, and a large meningioma in the right frontal region was removed. The tumor was benign.

The patient's outlook for life is excellent. Interestingly enough, at subsequent roentgenologic examination the previously suspicious widening of the duodenal loop could not be confirmed. In the six months after the operation there was no sign or symptom of abdominal tumor.

CASE 4.—A woman aged 45 had pain in the right upper abdominal quadrant, associated with periods of epigastric distress and occasional nausea and vomiting. Cholecystograms revealed cholelithiasis. Cholecystectomy was carried out, but the pain in the right

upper abdominal quadrant persisted and became progressively more troublesome.

This patient was seen three months after cholecystectomy. At this time she had clear-cut signs of a tumor of the spinal cord—weakness of both lower extremities, loss of sensation in the legs, some degree of spasticity in the legs, Babinski's sign, and incontinence of bowel and bladder.

Study of the patient's history shows that it would have been quite clear to a neurologist or a neurosurgeon that she had signs and symptoms highly suggestive of tumor of the spinal cord at the time of cholecystectomy. The pain in the right upper abdominal quadrant was typical of the pain caused by irritation of a nerve root. It had begun a year or more before the operation and had been fairly persistent. It extended from the spinal column, at the level of the eighth thoracic vertebra, along the costal margin and into the right upper quadrant. This pain was markedly accentuated when the patient coughed or sneezed.

Another characteristic of the pain is almost pathognomonic of tumor of the spinal cord: The pain invariably awakened the patient from four to six hours after she had retired, and she often had to walk the floor for relief or finish the night's sleep by sitting up in a chair. One possible explanation for this rather typical manifestation of the pain of a tumor of the spinal cord is that the tumor displaces and stretches the sensory roots overlying it. Then, as the patient lies down, the intervertebral discs, no longer compressed by the patient's weight, balloon out, thus elongating the spinal axis. This elongation causes the sensory roots to be stretched even more, and pain results. When the patient gets up, the intervertebral discs support weight and collapse to some extent, with the result that the stretch on the sensory root is lessened and pain consequently is relieved.

Myelographic investigation with ethyl iodophenylundecylate (Pantopaque) was carried out, and a defect in the opaque column was noted at the level of the eighth thoracic vertebra (Fig. 7). At operation a meningioma, a benign tumor, was completely removed (Fig. 8). Figure 8 diagrammatically illustrates the operative observations. It indicates how the eighth thoracic sensory root was stretched by the underlying tumor.

This patient has made definite improvement in the use of her legs since the tumor was removed. However, at the time of this writing she was unable to walk. We hope that in another year or so she will have regained most of her muscle power. It is our opinion, however, that had the tumor of the spinal cord been recognized and removed at the time the gallbladder was taken out she would not have been incapacitated subsequently.

Comment on Case 4. — Roentgenologic examination of the spinal column of a patient suspected of having a tumor of the spinal cord may show changes of diagnostic importance early in the course of the tumor, before many definite clinical signs are present. Calcification within the spinal canal, such as accompanies about 10 per cent of meningiomas, may be seen. In some cases, widening of the intraspinal canal and pedicular erosion are evident. In the presence of "dumb-bell" tumors, which grow from the nerve root inside the dura out through the intervertebral foramen, the latter aperture is abnormally enlarged.

As has been said, the foregoing changes sometimes are demonstrable roentgenologically before the clinical signs and symptoms are far advanced. When these demonstrable changes are added to other suggestive observations, the diagnosis of tumor of the spinal cord can be substantiated.

CASE 5.—The patient was a man 60 years old. He had been in good health until April 1953. At that time he had first noticed pain in the lumbar area of the back, with extension into both flanks. In August he had become progressively constipated, and it was thought that the caliber of his stools was smaller. He required more and more laxatives to produce a bowel movement. A gastrointestinal roentgenographic series was done; during this period the abdomen became distended. The results of the gastrointestinal roentgenograms were not available, and so it is not clear whether or not a lesion was disclosed. Surgical abdominal exploration was carried out, but no demonstrable cause for the abdominal distention was found. The patient was treated with suction for paralytic ileus, and the distention was relieved in this way.

At the time we saw him, in October 1953, he was unable to walk because of ataxia and marked weakness of the legs and ataxia. He was incontinent of bowel and bladder. Exami-

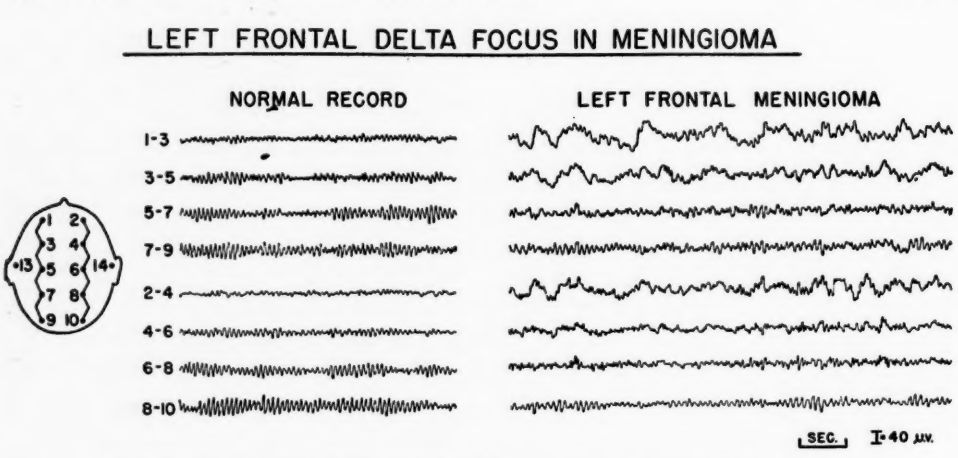


Fig. 5.—Electroencephalogram in Case 3, showing left frontal dysrhythmia.

nation revealed paresis of both lower extremities extending up to the eighth thoracic level and a sensory loss extending up to the same level. It was evident that this man had compression of the spinal cord. Roentgenologic examination of the spinal column revealed a destructive process involving the ninth and tenth thoracic vertebrae and a paravertebral soft tissue mass in this region.

At operation, compression of the spinal cord by caseous material, which proved to be of tuberculous origin, was observed. The caseous material was removed, so as to decompress the spinal cord. It is possible that this patient will regain the use of his legs, but it may be a year or more before he has the strength to walk.

Comment on Case 5. — Again, in this case, we are sure that the diagnosis of a lesion of the spinal cord could have been made before paralysis developed and an unnecessary abdominal operation carried out. The pain in the back was typical; it was aggravated by cough and sneeze and straining, and it awakened the patient at night. Constipation may accompany a lesion of the spinal cord—the patient avoids a bowel movement because the straining involved is painful. Then, as compression of the spinal cord increases, pathways concerned with innervation of the bowel and bladder become involved. At times, abdominal distention is associated with lesions of the spinal cord. This is true particularly when the damage to the cord is acute, as in cases of injury.

Had a roentgenogram of the spinal column been made in August, at the time of the abdominal operation, the destructive lesion would doubtless have been apparent.

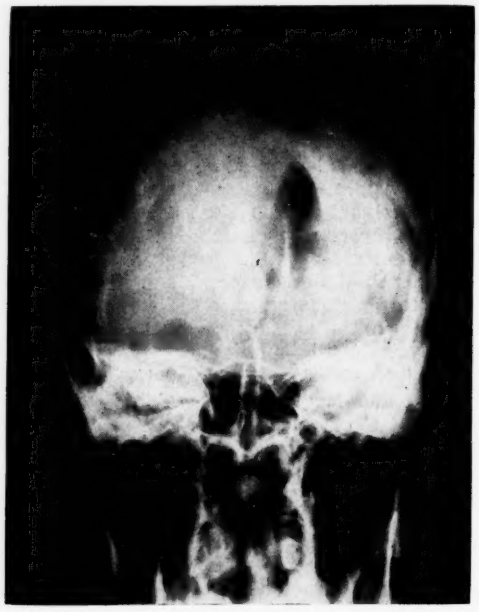


Fig. 6.—Ventriculogram, same case, indicating shift of the anterior portion of the lateral ventricles and the third ventricle to the right.

If the compression of the spinal cord had been relieved at that time, the patient would not have become paralyzed.

CASE 6.—A man aged 56 had undergone gastric resection for a Grade 2 small cell carcinoma two years prior to the time we saw him. He was examined because of pain in the right hip. The pain was severe and incapacitating, and it troubled him even at rest. Roentgenologic examination of the stomach and the site of anastomosis suggested local recurrence of the malignant process. The results of gastroscopic examination, however, were negative. Roentgenologic examination of the lumbosacral area of the spinal column gave negative results, but roentgenograms of the right hip showed some sclerotic changes, which could be interpreted as metastatic involvement. It was felt that the pain was most probably due to metastasis. Because of the severity of the pain, the patient insisted that something be done. Neurosurgical consultation was requested, with chordotomy in mind.

When the patient was questioned, it was learned that he had some degree of backache and pain radiating into the sciatic distribution, down to the ankle. The results of sciatic nerve-stretching tests were positive; the pain was accentuated by bending, coughing and sneezing. The attack had come on insidiously two months before, without any known trauma. However, it was learned that the patient had had similar, less severe and less prolonged attacks five years earlier.

Fig. 7.—Pantopaque myelogram in Case 4, showing defect in the opaque column on the right side at the level of the eighth thoracic vertebra. The defect is characteristic of an intradural, extramedullary tumor.

Fig. 8.—Sketch of operative observations in a case similar to Case 4 [reproduced, with permission of the publishers, from: Camp, J. D.: Contrast Myelography, M. Clin. North America 25: 1067-1102 (July) 1941].

It was concluded that the pain might well be due to a protruded intervertebral disc. Pantopaque myelographic study revealed a defect at the fourth lumbar interspace (Fig. 9). At operation a ruptured intervertebral disc was removed. Subsequently the patient was completely relieved of his pain.

COMMENT

Each of the foregoing 6 cases has these 2 points in common: (1) as far as the general surgical diagnosis is concerned, the condition present was what has been called "borderline," the signs and symptoms being atypical in some respects; (2) each of the patients had presented, at the time of the general surgical operation,

Fig. 9.—Pantopaque myelogram, indicating a defect at the fourth lumbar interspace on the right side in Case 6. The defect is typical of that produced by a protruded intervertebral disc.

signs and symptoms which, if not conclusive, were certainly suggestive of involvement of the central nervous system.

The lesson contained in these cases is clear: When the general surgeon sees a patient in whom a cardinal symptom or sign is lacking or atypically manifested, he must reevaluate the condition at hand from the standpoint of neurologic disorder, particularly if other signs or symptoms, not usually typical of the condition thought to be present, are detected. The neurologic or neurosurgical colleague of the general surgeon has many tools—myelography, carotid arteriography, electroencephalography roentgen rays, air studies and the like—which can be employed readily, together with the neurologic examination, without undue risk or delay.

Conversely, the neurologic surgeon has much to learn from the general surgeon. When the liaison between the two is good, the accumulated skills and knowledge of both cannot but redound to the welfare of the patient and enhance diagnostic acumen in the fields of both general surgery and neurosurgery.

SUMMARY

The authors report 6 cases of "borderline" neurologic lesions easily confused, by reason of their equivocal signs and symptoms, with lesions unrelated to the nervous system. They emphasize the fact that a better liaison between the general surgeon and the neurologist is needed, in the best interests of the patient, to prevent errors in diagnosis and delay in treatment. It is recommended that the general surgeon, when confronted with signs and symptoms indicative but not pathognomonic of a condition in his own field, consult the neurologist and avail himself of the many advantages provided by neurologic diagnostic aids — myelographic, carotid, arteriographic and electroencephalographic examination, air studies, etc. The authors point out that the neurosurgeon has much to learn also from the general surgeon, and that a closer relation between them would give the patient the benefit of their combined knowledge and experience.

RÉSUMÉ

Les auteurs rapportent six cas d'affections neurolgiques qui paraissent n'avoir aucun rapport avec le système nerveux, eu égard à des signes et symptômes équivoques, qui font penser à d'autres affections. Ils insistent, pour éviter des erreurs de diagnostic et de retard dans le traitement, sur une meilleure collaboration entre le chirurgien de culture générale et le neurologue. Il est recommandé au chirurgien de culture générale, lorsque le malade ne présente pas une symptomatologie pathognomonique, de consulter un neurologue et de faire une myélographie, une artériographie carotidienne, une électro-encéphalographie, une ventriculographie etc.

Les auteurs signalent que le neurochirurgien doit également tenir compte de l'expérience du chirurgien de culture générale. Cette collaboration entre les deux spécialistes ne pourra qu'être utile au malade.

RIASSUNTO

Gli autori riportano 6 casi neurologici che non vennero riconosciuti perchè le loro manifestazioni cliniche non furono chiare e le lesioni non erano in relazione col sistema nervoso. Ciò li porta ad insistere sul fatto che, nell'interesse dei pazienti, è necessaria una più stretta collaborazione fra chirurgo e neurologo ai fini di una diagnosi corretta e di una pronta terapia. Il chirurgo, cioè, ogni volta che si trova dinnanzi a sintomi non del tutto caratteristici di una malattia chirurgica, deve consultare il neurologo, in modo da potersi avvalere dei molti vantaggi offerti dai mezzi diagnostici neurologici, quali la mielografia, l'arteriografia, l'elettroencefalografia, la ventricolografia, ecc. L'autore sottolinea il fatto che il neurochirurgo ha molto da imparare anche dal chirurgo generale, sicchè una puú stretta collaborazione fra loro può offrire ai pazienti il beneficio della loro congiunta esperienza.

ZUSAMMENFASSUNG

Die Verfasser berichten über sechs neurologische Grenzfälle, die auf Grund ihrer zweideutigen klinischen Zeichen und Symptome leicht mit Erkrankungen verwechsel werden können, die in keiner Beziehung zum Nervensystem stehen. Es wird betont, dass zur Verhütung diagnostischer Irrtümer und zur Vermeidung einer Verzögerung in der Behandlung eine enge Zusammenarbeit von Chirurg und Neurologen notwendig ist, und dass auf diese Weise den Interessen des Kranken am besten gedient wird. Der Allgemeinchirurg wird angeregt, in Fällen, wo das klinische Bild zwar auf eine in sein Gebiet fallende Erkrankung hinweist, aber nicht pathognomonisch ist, einen Neurologen zu Rate zu ziehen, und sich der reichen Vorteile der neurologischen Diagnostik (Karotisuntersuchung, mysolographische, arteriographische, elektroenzephalographische und pneumoenzephalographische Verfahren) zu Nutze zu machen. Umgekehrt, kann auch der Nervenchirurg viel vom Allgemeinchirurgen lernen, und eine enge Zusammenarbeit beider gibt dem Kranken die Möglichkeit, von ihrer gemeinsamen Erfahrung und Kenntnis zu profitieren.

RESUMEN

Los autores comunican 6 casos de lesiones neurológicas incipientes que a causa de síntomas y signos equívocos son facilmente confundibles con lesiones no relacionadas al sistema nervioso central. Se hace notar el hecho de la necesidad de cooperación entre el cirujano general y el neurólogo para beneficio del paciente, para prevenir errores diagnósticos y retraso en el tratamiento. Se recomienda que el cirujano general ante signos y síntomas no patognómonicos indicadores de padecimiento comprendido en su campo de acción, debè consultar al neurólogo proveerse de los muchas ventajas que aportan los coadyuvantes diagnósticos de la neurología, exámenes mielográficos, arteriográficos carotídeos, electroencefalográficos, insuflaciones, etc. Los autores señalan que el neurocirujano tiene mucho que aprender también del cirujano general y que mientras más estrecha sea esta relación el beneficio será mayor para el paciente debido a la esperiencia y conocimiento combinados.

Transabdominal Anterior Discographic Examination

JOHN I. PERL, M.D., F.A.C.S.

CHICAGO, ILLINOIS

THE diagnostic value of discographic examination, the technic of which was first described by Lindblom,[1] was supported by the studies of Erlacher,[2] Witt,[3] Gardner and his associates,[4] Wise and his co-workers,[5] Cloward and Buzaid[6] and others.[7] It is the only procedure that permits roentgen visualization of the size and configuration of the nucleus pulposus. It is also the only one that permits recogtion of the various stages of degeneration of the nucleus, as well as the terminal changes represented by tear or rupture of the annulus, with prolapse or herniation of the pulpous material.

Although it is established that discographic study yields a far greater percentage of correct diagnoses than do other, more conventional diagnostic procedures either singly or in combination, its more frequent employment is hampered partly by the technical difficulty of the procedure and partly by the clinical sequelae inherent in the transdural and epidural injection of radiopaque solution into the disc.

The standard posterior discographic or nucleographic investigation is performed as follows: A spinal needle of 18 to 20 caliber is introduced between the spinous processes at the desired lumbar level and advanced, either transdurally or epidurally toward and to the intervertebral disc to be examined. After the proper position of this needle has been ascertained by roentgenograms, a second needle, of much smaller caliber (22-26 gauge), is inserted through the lumen of the previously placed needle until the tip of the second needle reaches the approximate center of the disc. Additional roentgenograms are taken to verify the position of the second needle within the disc substance. A water-soluble opaque material is then injected through the second needle, and anteroposterior and lateral films are taken. Usually two and occasionally three discs are examined in this manner. It is obvious that the procedure must be time-consuming even in experienced hands.

The purpose of this communication is to present the technic of transabdominal anterior discographic examination. With an open abdomen this is a simple procedure. It can be speedily and safely performed upon patients with chronic backache (a very large group) who, for various other reasons, are undergoing operations on the pelvis or the lower part of the abdomen.

During investigative work in connection with transabdominal supplemental spinal analgesis,[8] I made a series of anterior disc injections to determine whether any leakage of the pulpous nucleus occurs along the puncture tracts of the spinal needles employed with their various calibers. The ease with which these injections were accomplished prompted the idea that anterior or transabdominal discographic methods could be used to advantage during laparotomies. Subsequently a total of 37 anterior disc injections were performed on 15 unselected patients. Injections were made into the disc L 3—4 in 6 cases, into the disc L 4—5 in 14 cases and into the lumbosacral disc in 12 cases. During a pneumonectomy injection of contrast medium into 5 intervertebral discs was done for clinical study. These are also in-

From the Department of Surgery, Augustana Hospital, Chicago.

Supported by funds from the Nelson M. Percy Research Foundation.

Submitted for publication Nov. 18, 1953.

cluded in the series under discussion.

The opaque solution used was Diodrast in concentrations of 35 to 70 per cent. History of backache and other pertinent information is presented in the accompanying table.

Indications.—In patients whose histories include complaints of chronic recurrent backache and who are undergoing operations in the pelvis or the lower part of the abdomen, transabdominal discographic investigation can be performed with advantage as a diagnostic procedure.

Contraindications.—The procedure should not be employed in the presence of (a) infection of the peritoneal cavity or its contents, (b) malignant disease with metastases along the large vessels or (c) marked obesity.

Procedure.—The equipment used for disc injections consists of two Sana-lok syringes of 3 cc. capacity, each loaded with Diodrast and armed with a standard curved tonsil needle. The long-shafted tonsil needle has a 23-gauge tip ½ inch (1.2 cm.) long, which, when fully inserted through the annulus, penetrates into the substance of the pulpous nucleus, thus depositing the opaque solution at the same depth in every disc into which an injection is made.

The Sana-lok syringe has a twofold advantage: first, it has rings for the fingers —this assures a firm, nonslip grasp, which is important when appreciable pressure has to be employed while the resilient nucleus of the disc is injected. Second, it has a short, smooth metal plunger, which slides within the barrel of the syringe with far less friction than the full length cylindric ground-glass plunger. Both of these features are of advantage

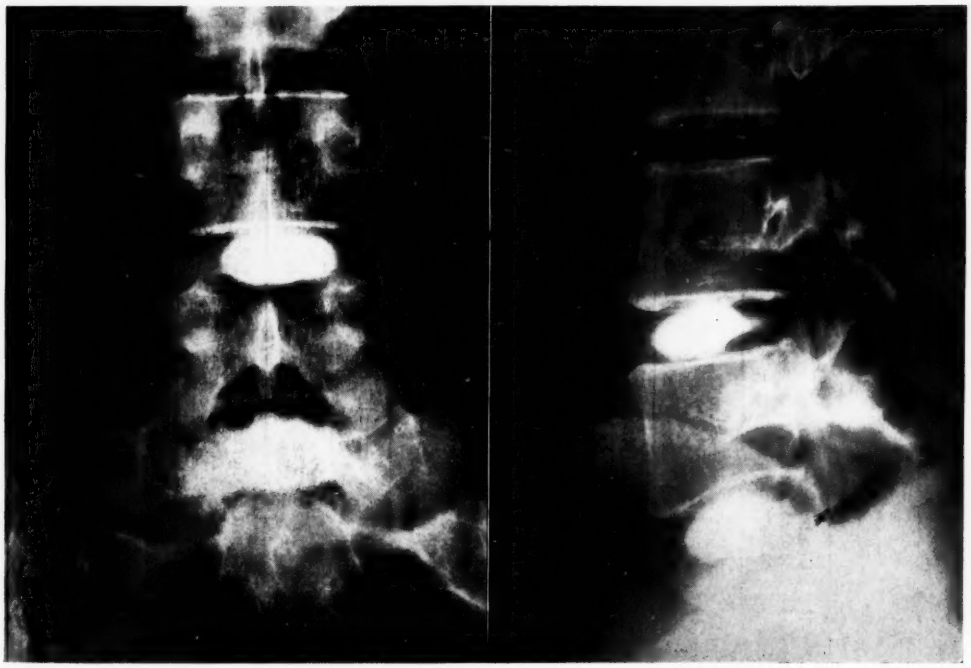


Fig. 1.—Discogram of a patient with history of low backache for the last three months preceding the operation. Note the normal square nucleus in the lumbosacral disc and the oval nucleus with a single posterior branch at disc L 4-5.

Data on 15 Transabdominal Anterior Discographic Examinations

Case	Patient's Sex and Age	Operation	Discographic Study		Roentgen Data	History of Low Backache			Duration
			Disc	Diodrast, Cc.		Lumbago	R	Radiation L	
1	F 54	Bilateral salpingo-oophorectomy; hysterectomy	L 4-5	*2	Branched nucleus with posterior herniation	+	0	+	Intermittent 15 years
			L 5-S 1	*1	Simple branched nucleus				
2	F 41	Right oophorectomy; total hysterectomy	L 4-5	*2	Simple lobed nucleus	+	0	+	Intermittent 14-15 years
			L 5-S 1	*2.5	Multiple-branched nucleus				
3	F 39	Bilateral salpingo-oophorectomy; total hysterectomy	L 4-5	*2.5	Multiple-lobed nucleus	+	+	0	Periodic 21 years
			L 5-S 1	*3	Simple-lobed nucleus				
4	F 29	Total hysterectomy	L 3-4	*2	Normal globular nucleus	0	0	0	0
			L 5-S 1	*1	Normal globular nucleus				
5	F 47	Right salpingo-oophorectomy; total hysterectomy	L 3-4	**1	Flattened, lobulated nucleus with anterior herniation	+	0	0	5 months
			L 4-5	**2	Normal square nucleus				
6	F 33	Repair of ventral hernia	L 3-4	**1	Normal oval nucleus	+	0	0	Intermittent "for years"
			L 4-5	**2	Normal square nucleus				
			L 5-S 1	**1	Normal oval nucleus				
7	F 25	Right oophorectomy	L 4-5	**1	Oval nucleus with small single posterior branches	0	0	0	3-4 months
			L 5-S 1	**1	Normal square nucleus				
8	F 44	Left salpingo-oophorectomy; total hysterectomy	L 4-5	**1	Normal globular nucleus	+	0	0	5 years
			L 5-S 1	**2	Multiple-lobed nucleus				
9	F 60	Bilateral oophorectomy	L 4-5	**0.5	Normal globular nucleus	0	0	0	0
			L 5-S 1	**2	Normal oval nucleus				

10	F 57	Bilateral salpingo-oophorectomy; total hysterectomy	L 5-S 1	**1	Normal bivalve nucleus	0	0	0	0
			L 3-4	**1	Multiple-branched nucleus with posterior herniation				
			L 4-5	**2	Multiple-lobed nucleus				
11	M 65	Repair of venyral hernia	L 3-4	**0.2	Extradiscal injection (osteophyte)	+	+	0	3 years
			L 4-5	**1	Multiple-branched nucleus with posterior herniation				
			L 5-S 1	**1.5	Multiple-branched nucleus with postoperative prolapse				
12	F 41	Total hysterectomy	L 4-5	**1	Normal oval nucleus	0	0	0	0
			L 5-S 1	**2	Normal oval nucleus				
13	F 37	Breast amputation; bilateral oophorectomy	L 3-4	**1	Normal bivalve nucleus	+	0	0	2 years
			L 4-5	**1.5	Flattened lobulated nucleus with posterior herniation				
			L 5-S 1	**1.5	Lobed nucleus				
14	M 70	Right pneumonectomy	T 6-7	**1	Flattened lobed nucleus	+	0	0	Many years
			T 7-8	**1	Flattened lobed nucleus				
			T 9-10	**0.3	Large osteophyte; small flat anterior nucleus				
			T 10-11	**1	Flattened lobed nucleus				
			T 11-12	**1	Normal bivalve nucleus				
15	F 34	Right salpingo-oophorectomy	L 3-4	**2.5	Lobed nucleus with incipient posterior herniation	0	0	0	0
			L 4-5	**1.5	Normal square nucleus				
			L 5-S 1	**1.5	Normal square nucleus				

*35 per cent Diodrast
**70 per cent Diodrast

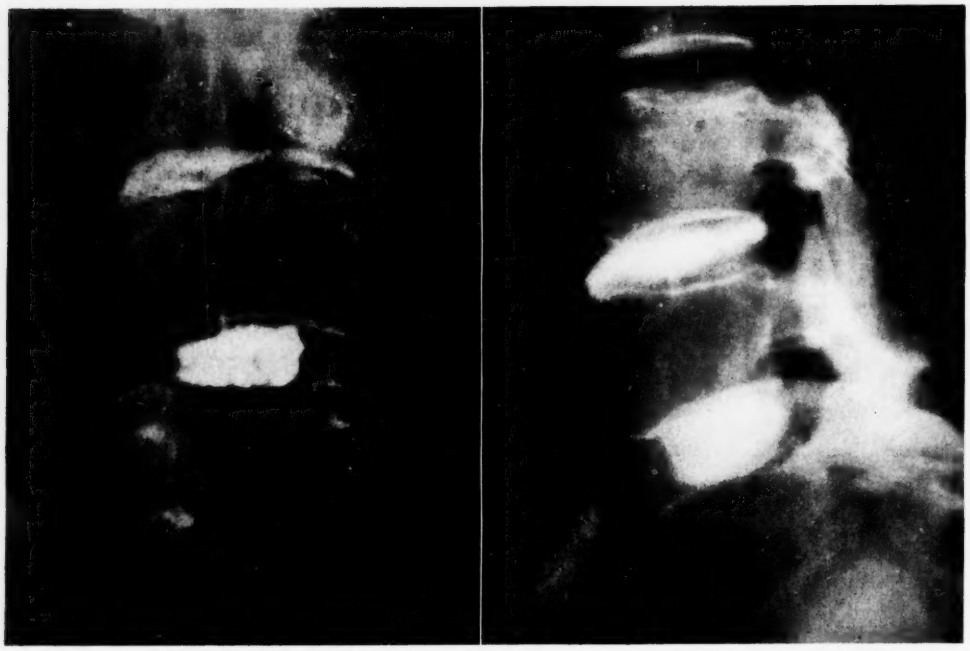


Fig. 2.—Discogram of a patient with a history of low backache without sciatic radiation for five months before operation. Note the flattened multilobulated nucleus with anterolateral and posterior herniation at Disc L 3-4. The disc at L 4-5 shows a normal square nucleus.

when the near-saturate solution of opaque medium is injected through a small-caliber needle into a substance that possesses considerable intrinsic tension.

If anterior discographic study during laparotomy is contemplated, a midline incision should be preferred. This approach, with the muscular relaxation and quiet abdomen associated with spinal analgesia, is ideal for good exposure of the pelvic organs and the distal lumbar segments. In patients of average or normal physical proportions, incision between the navel and the symphysis readily allows injection of the fluid into the discs at L 4=5 and L 5=S 1. If the patient is short and squatty, or if it is desired to reach the disc at L 3=4 or a higher segment, the incision must be extended along the left side of the umbilicus or even ½ inch (1.2 cm.) above it.

After routine exploration, the patient is placed in the Trendelenburg position to allow the intestines to slide into the upper part of the abdomen where they are confined with packs and retractors.

Unless the patient is markedly obese, there is no difficulty in visualizing the aorta, or in defining its contour by palpation. In like manner, the slightly elevated annulus of the disc, which gives a sensation of elastic rebound to the palpating finger, can be readily differentiated from the much wider, somewhat concave, nonelastic rigid vertebral body.

As the disc is located, the nail of the palpating finger is firmly anchored to the convexity of the elastic annulus and held in position there until the needle is inserted and the injection completed.

The needle should never be advanced forcibly. The disc margin (the annulus fibrosus) will always permit easy introduction of the needle unless it is shielded

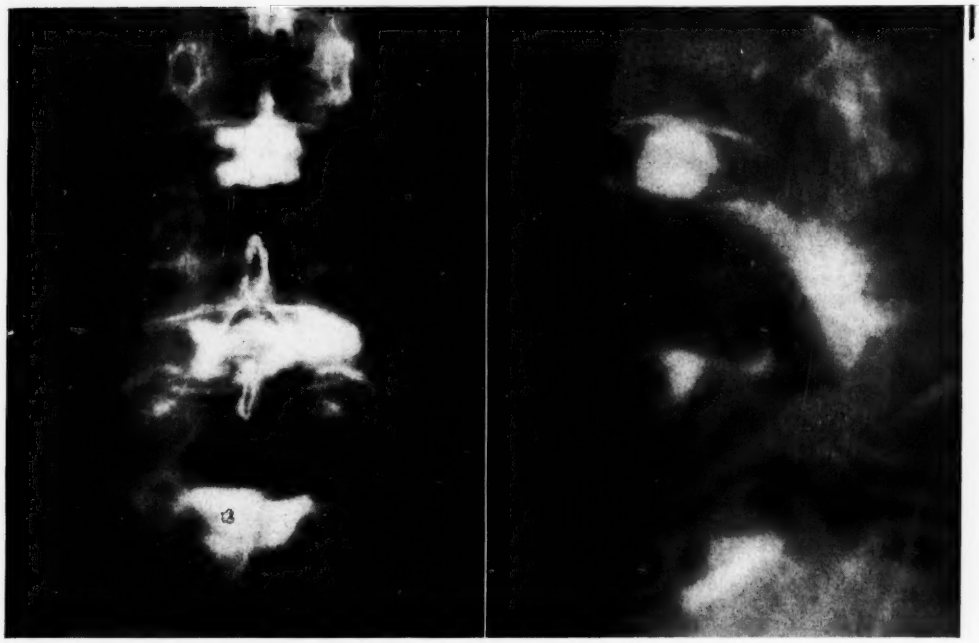


Fig. 3.—Discogram of a patient with no history of low backache. Here the three lower lumbar discs treated by injection show various morphologic stages of the nucleus pulposus. At the disc at L 3-4 fourth lumbar levels there is a bivalved, globular normal nucleus. At the disc L 4-5 the nucleus is multibranched, with posterior prolapse into the spinal canal. The nucleus of the lumbosacral disc is multilobulated, representing a type liable to progress to herniation.

by an osteophyte. Any hard resistance to penetration of the needle indicates that the needle has met bone, or the cartilaginous endplate of the vertebral body, neither of which should be forced or traumatized.

As a rule, the lumbosacral disc is first identified below the bifurcation of the large vessels. The full length of the needle is inserted into its substance, and as much Diodrast is injected as the disc will take with moderate pressure.

If there is a high bifurcation of the aorta and the inferior vena cava, the disc at L 4=5 might also be injected in the midline. In the majority of cases, however, it is injected on the left anterolateral sector of the spine alongside the aorta, while the index finger guards and slightly displaces the vessel toward the midline. The disc L 3=4 can be injected in a similar manner.

As soon as the needle penetrates the annulus, moderate pressure should be exerted upon the plunger of the syringe. This serves to counteract the intrinsic tension within the disc, which might force pulpous fluid back into the lumen of the needle, occluding it. Even the normal disc will accommodate approximately 0.5 to 1 cc. of fluid; therefore, resistance to the injection so strong that no fluid can be deposited implies either that the needle is occluded with precipitate or that its tip is embedded in the endplate of the vertebral body. Under such circumstances the needle should be withdrawn and its patency tested before it is reintroduced at a slightly different angle.

Immediately after the injections, roent-

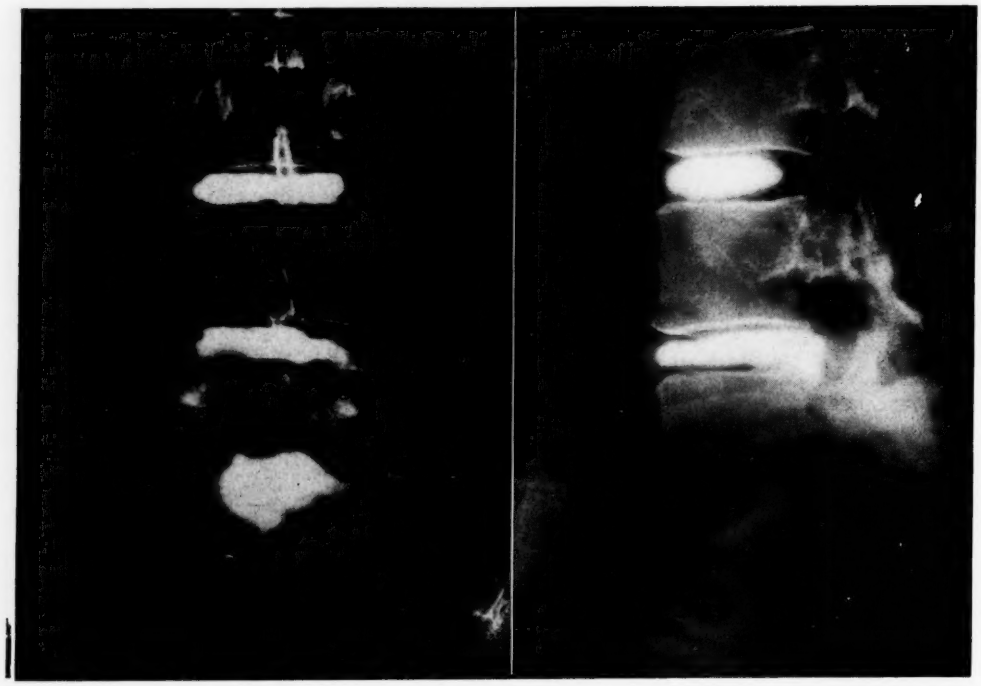


Fig. 4.—Discogram of a patient with a history of a short episode of low backache two years before discographic study. The discogram shows a normal nucleus at disc L 3-4, a flattened, lobulated nucleus with posterior prolapse at disc L 4-5, and a multibranched nucleus at the lumbosacral disc.

genograms are taken in the anteroposterior and lateral positions and the contemplated operation is carried through to completion (Figs. 1 to 5).

COMMENT

A small series of transabdominal discographic examinations performed during pelvic operations has impressed me with the ease and speed with which injections of contrast medium into the lower lumbar discs can be accomplished. Both advantages are in marked contrast to the consumption of time and the repeated roentgen check-ups required before the opaque material can be injected into the pulpous nucleus by the posterior discographic technic.

No postoperative complaints referable to the discographic procedures were encountered in the present transabdominal series. This is understandable, since trauma to the soft tissues of the back and the periosteum of the spine by multiple needle stabs, and the repeated puncture transfixions of the epidural space or the dural sac—unavoidable features of the posterior epidural or transdural discographic study—are avoided by anterior injection of the contrast medium into the disc.

That transabdominal discographic examination can be performed only during a laparotomy is admittedly a limiting factor. Nevertheless, it gives the general surgeon and the gynecologist an excellent opportunity to investigate the status of the lower lumbar discs in patients with chronic low backaches, who for other rea-

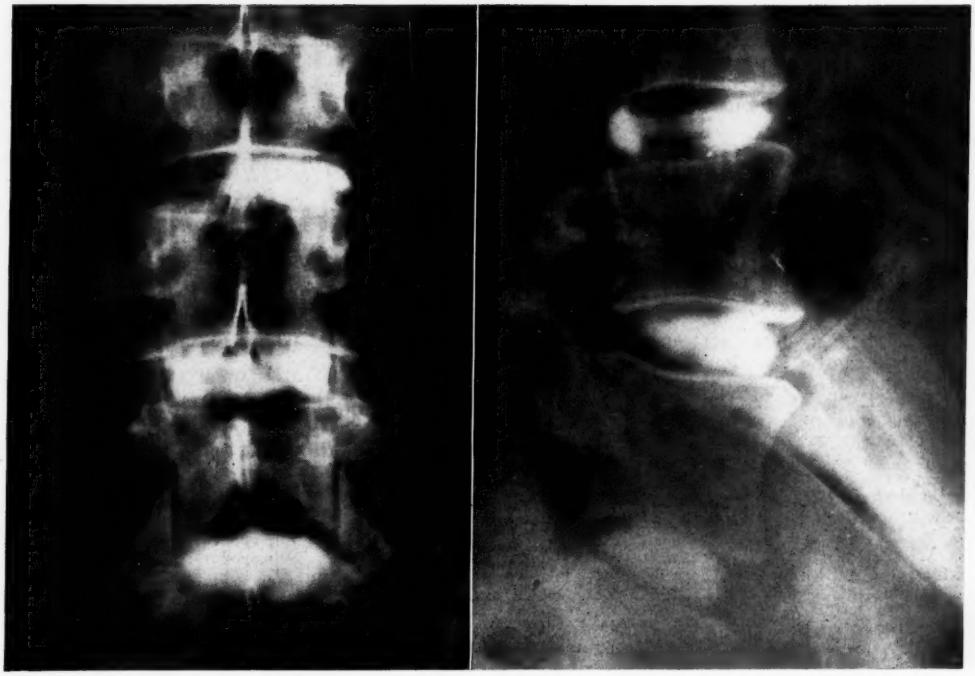


Fig. 5.—Discogram of a patient who, even upon the closest interrogation, could not recall an incident of low backache. She admitted severe cramps with pain around the waist during the first days of her menstrual periods. At the third and fourth lumbar level the discogram shows a flattened, multibranched nucleus with a mushroom-shaped, posteriorly extending deformity representing a clinically silent and structurally still incomplete rupture of the posterior segment of the annulus. The discs at L 4-5 and first sacral level present normal configuration.

sons undergo operations of the pelvis or the lower part of the abdomen.

SUMMARY

A technic for performing transabdominal anterior discography as an addendum to lower abdominal operations is presented with the report of observations in 15 unselected cases.

ZUSAMMENFASSUNG

Es wird eine Technik der röntgenologischen Kontrastdarstellung von Zwischenwirbelscheiben von vorn auf transabdominalem Wege gelegentlich von Unterleibsoperationen dargestellt und über die Beobachtungen an 15 nicht ausgewählten Fällen berichtet.

SUMARIO

O autor apresenta uma técnica para a realização da discografia anterior transabdominal afim de completar operações abdominais inferiores, comunicando 15 observações de casos não selecionados.

RESUMEN

Se presenta una técnica para llevar a cabo discografía anterior transabdominal como un suplemento de las operaciones de vientre inferior. Se comunican 15 observaciones de casos no seleccionados.

RIASSUNTO

Viene descritta la tecnica per la discografia anteriore transaddominale, da impiegarsi negli interventi sui quadranti inferiori dell'addome.

REFERENCES

1. Lindblom, K.: Diagnostic Puncture of Intervertebral Disk in Sciatica, Acta Orthop. Scand. 17:231, 1948. Technique and Results of Myelography and Disk Puncture, ibid. 34:324-330 (Oct.-Nov.) 1950. Discography of Dissecting Transosseous Ruptures of Disks in Lumbar Region, Acta Radiolog. 36:12-16 (July) 1951. Technic and Results of Diagnostic Disc Puncture and Injection (Discography) in Lumbar Region, Acta Orthop. Scand. 20:315-326, 1951.

2. Erlacher, P.: Clinical and Diagnostic Significance of Nucleography, Ztschr. f. Orthop. 79:273-278, 1950. Direkte Kontrastdarstellung des Nucleus Pulposus, Zugleich ein Beitrag zur Pathologic der Bandscheibe, Ztschr. f. Orthop. 80:40-57, 1950. Nucleography, J. Bone & Joint Surg. 34-B:204-210, 1952.

3. Witt, A. N.: Praktische erfahrungen mit der nukleographic, Ztschr. f. Orthop. 80:57, 1951.

4. Gardner, W. J.; Wise, R. E.; Hughes, C. R.; O'Connel, F. B. Jr., and Weiford, E. C.: X-ray Visualization of the Intervertebral Disc, with Consideration of Morbidity of Disc Puncture, Arch. Surg. 64:355, 1952.

5. Wise, R. E.; Gardner, W. J.; Hughes, C. R., and Weiford, E. C.: X-ray Visualization of the Intervertebral Disk, Modern Med. 20:104-112, (June), 1952. Wise, R. E., and Weiford, E. C.: X-ray Visualization of Intervertebral Disk, Cleveland Clin. Quart. 18:127, 1951.

6. Cloward, R. B.: Anterior Herniation of Ruptured Intervertebral Disc, with Comments on Diagnostic Value of Discogram, Arch. Surg. 64:457-463, 1953. Cloward, R. B., and Buzaid, L. I.: Discography: Technique, Indications and Evaluation of the Normal and Abnormal Intervertebral Disc, Am. J. Roentgenol. 68:552-564 (Oct.) 1952.

7. Dupuis, P. V.; Delbeke, P.; De Haene, R., and Lassoie, A.: Les enseignements de la discographie, Acta Orthop. Belg. 18:309-316 (Dec.) 1952. Gleason, P. G.: The Technic of Discography, U. S. Armed Forces M. J. 3:1831-1837 (Dec.) 1952. Hoen, T. I.; Druckemiller, W. H., and Cook, A. W.: Injection of Lumbar Intervertebral Disks, U. S. Armed Forces M. J. 2:1067-1074 (July) 1951. Romanus, R., and Yden, S.: Discography in Ankylosing Spondylitis, Acta Radiol. 38:431-439 (Dec.) 1952. Smith, G., and others: Presentation at A.M.A. convention, 1951.

8. Perl, J. I.: Transabdominal Supplementary Spinal Analgesia, J. Internat. Coll. of Surg. 20:343-349 (Sept.) 1953.

The art of medicine was old before universities were founded, but the science of medicine is younger than all but the most recent of them. We often picture the development of medical science as the slow building up or deduction of knowledge from experience accumulated over many hundreds of years; but the fact is exactly the reverse. Medicine, in common with other sciences, began a *priori* with inductive reasoning from general assumptions about the nature of life and disease, and only gradually came to use what are now called scientific methods of experiment and observation. The scientific outlook, which has recourse to inquiry rather than assumption, appeared with the Renaissance, and inspired men like Vesalius and Harvey to spurn tradition and seek for truth.

—*Ogilvie*

A New Method for Anesthetizing the Inferior Alveolar Nerve*

ZIYA CAMAL B. AKSOY, M.D., D.S., F.I.C.S.†

ISTANBUL, TURKEY

IT is known that the mandibular rami may differ in length and width. Using a specially devised compass in normal subjects, I arrived at the conclusion that the space between the anterior and posterior borders of the rami differs widely. According to these measurements, except in the presence of pathologic conditions (dwarfism, gigantism or acromegaly, for instance), the difference between the widest ramus and the narrowest is about 18 mm.

According to various studies on the rami of naked mandibles,** the average width of the rami at the level of the foramen mandibulae may range from 22 to 40 mm. Since it is outside the scope of this presentation, I shall not here consider why these variations occur.

Photographs of different mandibles have shown that, even in the same person, the left and the right rami may differ in length and width.

When anesthetic solutions are to be injected into the mandibular foramen and when the oral route is used, one must first determine the depth of the mandibular foramen from the anterior border by measuring, after which the anesthetic should be administered accordingly. As a matter of fact, from the aforementioned studies of naked mandibles, the space from the anterior border to the foramen varies approximately from 14 to 28 mm. When alcohol is to be injected the importance of these measurements becomes clear. Even when one employs extra-oral injection through the posterior border, such measurements are essential.

When injection is through the lower border, since the length differs, one may think it necessary to measure the depth of the foramen; it is known, however, that the mandibular foramen is continuous with the mandibular sulcus under the foramen; therefore no measurements may be used.

On the basis of these observations I undertook to devise an instrument that could be used on patients to measure the width of the mandibular ramus between the anterior and posterior margins at the level of the foramen (B. Aksoy's compass, Fig. 1). The compass consists of two legs, one curved and fixed and the other straight and movable. I measure this width by applying the fixed leg of the compass to the posterior border of the ramus on the skin below the ear, the movable leg being inserted through the mouth to the anterior border. The resulting measurements therefore include the soft parts.

From the readings on both sides of the movable leg one can determine the width in millimeters. This having been done, it is easy to estimate the depth of the foramen. Studies on both living and dead subjects have shown a definite relation between the depth of the foramen and the width of the ramus. It was therefore concluded that the distance of the foramen from the anterior border is three-fifths the width of the ramus.

The distance from the deepest part of the mandibular notch to the lower border, on a line parallel with the posterior

*Demonstration given at the International Dental Congress, London, England, July 24, 1952.

†Deceased. Formerly Director of the Institute of Oral Surgery, University of Istanbul.

**Nevin, M.: Length of Needle Insertion in a Mandibular Injection, Modern Dentistry 16, 1949.

Read at the Eighteenth Annual Congress of the United States and Canadian Sections, International College of Surgeons, New York, Sept. 13-17, 1953.
Submitted for publication April 24, 1954.

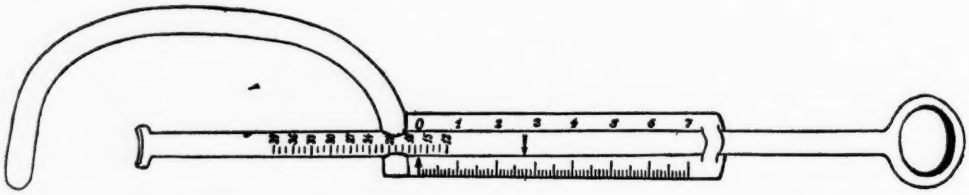


Fig. 1.—B. Aksoy's dividers for measuring the width of the rami in the living subject (see text for explanation).

border of the rami, differs also in different subjects. As has been mentioned, when the extra-oral lower border route is used, this longitudinal distance must be estimated to enable one to reach the foramen at the right point. This depth of the foramen from the lower border is also three-fifths of the measurement aforementioned. Thus it is clear that by measuring the depth and width of each ramus one can determine the exact location of the foramen by the following formulas: If F is used to show the depth of the foramen from the anterior border and L represents the length or width of the ramus, the formula is $F = \frac{3L}{5}$ (B. Aksoy's index). For instance, if the width of the ramus is 35 mm., the foramen is situated 21 mm. from the anterior border $\left(\frac{35 \times 3}{5} = 21\right)$.

According to this formula (B. Aksoy's index) I prepared a second type of compass (a modification of the first), with a second scale on the movable leg to show, in millimeters, the depth of the foramen from the anterior border, so that there is no need to calculate the depth. By measuring, with any instrument, the length of the ramus, and using the same formula, one can estimate the depth of the foramen from the anterior border. For instance, if the length of the ramus from the mandibular notch (incisura) to the lower border of the ramus is 55 mm., the foramen is situated 33 mm. from the lower border. $\left(\frac{3 \times 55}{5} = 33\right)$.

One can also measure the depth of the foramen from the posterior border by changing the formula thus: $\frac{2L}{5}$. If the width is 35 mm., the foramen is 14 mm. from the posterior border $\left(\frac{35 \times 2}{5} = 14\right)$.

Another idea that made intra-oral anesthesia very easy was the preparation of a mandrin (stilet). For anesthesia of the inferior alveolar nerve, using the oral route in order to plunge the needle to the determined depth, I prepared two types of mandrins to be used on curved needles. The mandrins are employed to determine the depth of the foramen from the anterior margin. They consist of metallic tubes (Fig. 2) to contain the needles and a vertical portion which is placed at the anterior border. I call this vertical piece "cursor-fix, syn. marking arm." On the end of the tube containing the needle there is a cap, which is used to fix the pavilion of the needle to the tube.

If every dental surgeon would make for himself this type of mandrin in his own laboratory, he could avoid buying ready-made mandrins. I advise the preparation of three mandrins, each of which has a tube 25 mm. long. The cursor should be fixed to the tube at different levels; on one of them 20 mm., on another 15 mm., and on the third 8 mm., from the distal tip. Through the tube of this mandrin I pass flexible and oxidation-proof needles 38 to 44 mm. long. Bending by hand will produce the length desired.

The second type of mandrin is more useful than the simple original version and more suitable for regulating the distance. The main difference is that in the second

type the vertical marking arm (locating lug) can be moved open downward by a screw, and consequently the point of the needle can be brought precisely to the desired millimeter of depth. It is possible to obtain similar mandrins with movable marking arms (syn. cursor mobile) from the commercial market.

B. Aksoy Method of Peroral Anesthesia of the Inferior Alveolar Nerve.—The patient is seated in a dentist's chair. The ramus on the side on which anesthesia is desired is measured with the B. Aksoy special compass (Fig. 1).* If this shows that the point of the needle should be at a depth of 24 mm. from the anterior margin, then, with the aid of one of the mandrins (the simple type or the one with the movable marking arm) the needle and the arm are adjusted to 24 mm., the needle and mardrin being mounted on a 2 cc. Record syringe.

I keep the patient with the mouth wide open and the commissura labialis pushed aside by a mouth inspection mirror. After ascertaining the midpoint on the pterygomandibular raphe and assuring myself that the mandrin is at the right level, I inject a few millimeters of the solution at the point where the periosteum and the marking arm are estimated to be 8 to 10 mm. from the anterior border, in order to anesthetize the lingual nerve. The needle is then inserted until the marking arm touches the anterior border, after which the necessary amount of alcohol or anesthetic solution is injected.

*As has been mentioned, the second type of compass shows both the width of the ramus and the depth of the foramen in millimeters. This type is the more useful, as it indicates the exact location of the foramen without calculation.

SUMMARY

When the intra-oral or extra-oral lower or posterior route is used for anesthesia of the inferior alveolar nerve, it is impossible to reach the exact site of the mandibular foramen without measuring the width or the length of the ramus in each case and on each side. Although anesthesia can be induced even when the solution is not injected at the exact site of the foramen, it is especially important that the exact site be reached when alcohol is employed. For this purpose the author has prepared a special compass to measure the width of the mandibular ramus and

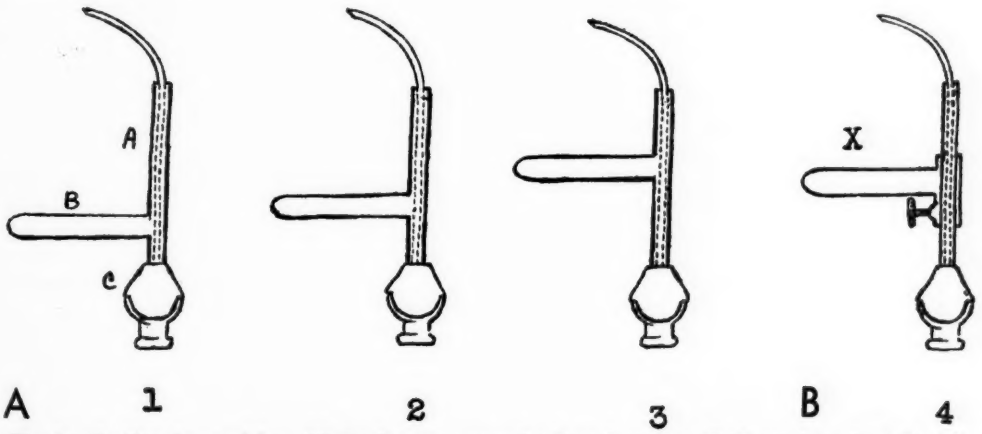


Fig. 2.—B. Aksoy's mandrins with fixed cursors carrying bowed needles. Each consists of a tube (*A*) through which the needles pass, a fixed cursor marking arm (*B*) vertical to this tube, and a cap (*C*) which is to be fixed upon the flaring end of the needle. The marking arm (*B*) is fixed upon the tube at point *1* at a distance of 20 mm., at point *2* at a distance of 15 mm., and at point *3* at a distance of 8 mm. from the distal tip. *B*, B. Aksoy's mandrin carrying a bowed needle and having a movable marking arm (*B*). This movable cursor can be fixed at the desired depth from the anterior margin by moving it up and down the tube.

has set up a formula to calculate the exact location of the foramen. He has also prepared special mandrins (stilets) through which the solution can easily be injected at the correct point. This new method of anesthetizing the inferior alveolar nerve by the intra-oral route is in current use at the Institute for Oral Surgery in Istanbul.

RÉSUMÉ

Il est impossible, lors d'anesthésie par voie extra ou intra-buccale du nerf dentaire inférieur, d'atteindre l'emplacement exact de l'orifice interne du canal dentaire inférieur sans mesurer sa longueur de chaque côté. Quoique l'anesthésie puisse être obtenue même si la solution n'est pas injectée à l'endroit précis de l'orifice, il est indispensable, pour les injections d'alcool, d'atteindre l'emplacement exact. Dans ce but, l'auteur a préparé, d'une part, un compas spécial qui peut mesurer l'étendue du nerf mandibulaire, et, d'autre part, une formule pour calculer l'emplacement de l'orifice interne du canal dentaire. Il a également confectionné un trocart à travers lequel il est facile d'injecter la solution à l'endroit désiré.

ZUSAMMENFASSUNG

Bei der Anästhesierung des unteren Alveolarnerven auf intraoralem oder extraoralem unterem oder hinterem Zugangswege ist es unmöglich, die Stelle des Foramen mandibulare genau zu treffen, ohne in jedem Fall und auf jeder Seite die Breite oder Länge des Ramus zu messen. Anästhesie lässt sich zwar auch erzielen, wenn e Lösung nicht genau in das Unterkieferloch eingespritzt wird, bei Alkoholinjektionen jedoch ist das Treffen der richtigen Stelle wesentlich. Zu diesem Zweck hat der Verfasser einen speziellen Zirkel konstruiert, mit dessen Hilfe man die Breite des Kieferastes messen und eine Formel zur Errechnung des genauen Sitzes des Unterkieferloches aufstellen kann. Ausserdem hat er Kanülen gebaut, mit denen sich die Lösung leicht an der richtigen Stelle einspritzen lässt. Das Institut für Orale Chirurgie in Istanbul bedient sich jetzt dieser neuen Methode zur intraoralen Anästhesie des unteren Alveolarnerven.

RESUMEN

Cuando se utiliza la vía posterior ó extraoral inferior en la anestesia del nervio dentario inferior, es imposible alcanzar el sitio exacto de foramen mandibular sin medir la amplitud y longtiud de la rama en cada caso y en cada lado. Aun cuando puede obtenerse anestesia cuando la solución no se inyecta en el sitio del foramen; empleando inyecciones de alcohol es esencial el lograr el sitio correcto. Para este proposito el autor ha preparado un compás especial que puede medir la amplitud de la rama mandibular y establecer una fórmula para calcular el sitio excato del foramen mandibular. Tambien ha preparado estiletes con los cuales se puede inyectar facilmente la solución en el sitio correcto. Este método nuevo para anestesia intraoral del nervio dentario inferior esta siendo usado actualmente en el Instituto de Cirugía Oral en Estanbul.

RIASSUNTO

Quando, nel praticare l'anestesia del nervo alveolare inferiore, si segue la via intra- o extra-orale o posteriore, è impossibile raggiungere la sede esatta del foro mandibolare senza misurare la larghezza e la lunghezza del ramo mandibolare in ogni caso e da ogni lato. Per quanto l'anestesia possa essere ottenuta anche quando la soluzione anestetica non sia stata iniettata nella sede esatta del foro, allorchè si pratichino iniezioni alcooliche è d'importanza essenziale che tale sede sia esattamente raggiunta. Allo scopo l'autore ha preparato uno speciale compasso che misura la larchezza del ramo mandibolare e fornisce una formula capace di indicare la sede esatta del foro mandibolare Egli ha anche preparato aghi attraverso cui si può facilmente iniettare la soluzione nel punto giusto.

Spontaneous Intracerebral Hematoma

WILLIAM R. CHAMBERS, M.D., F.A.C.S., F.I.C.S.

ATLANTA, GEORGIA

SPONTANEOUS (nontraumatic) intracerebral hematoma is now being widely recognized as a type of "stroke" calling for surgery in selected cases. Aring[1] has announced, "The only treatment of any avail is surgical removal." Odom, Bloor and Woodhall[2] have stated, "All of the resources of the neurosurgical armamentarium should be used toward the operative relief of patients with an intracerebral hematoma." Grant,[3] Scott,[4] Browder,[5] and others, after study of large series of cases have all favored a well-considered operative approach.

The problem is a pressing one, since Scott[4] has pointed out that "stroke" has risen to fourth place among causes of death. Falconer[6] has estimated that 80 per cent of those who suffer from intracerebral clot die within two to twenty-four hours of onset and has declared that cerebral hemorrhage is the principal killing disease of the nervous system. He adds, "Many of these deaths undoubtedly could be prevented." De Takats and Graupner[7] recorded the mortality rate at 77 per cent.

The problem is not simply the reduction of mortality. The question most frequently posed by the family is whether, if the patient recovers, he will be paralyzed or mentally impaired. The family physician wants to know whether or not a recurrence can be expected in a very short time if the patient survives. Aring[1] has pointed out that recurrence is not the rule. Others have shown they occur in only about 10 per cent. Margolis and his associates[8] have demonstrated in at least some of these patients that the hemorrhage is the result of a small angiomatous malformation, which is usually destroyed either by the hemorrhage or by surgical intervention.

The outlook as to lasting morbidity is a more distressing problem. Some have wished to predicate selection for operation on a basis of age. Experience has shown that arbitrary selection of only patients below 50 years of age is grossly unfair to many victims of the disease, because, while the age factor is certainly relevant, it is not of primary importance. Five other considerations are at least equally important. The first is whether or not the patient is initially in coma. The vast majority of patients with spontaneous intracerebral bleeding have an apoplectic onset with coma; the prognosis is better for those who do not. The second is whether or not those who have an onset with coma begin to show some evidences of neurologic improvement within twenty-four to forty-eight hours. Browder and Corradini[5] have pointed out the favorable indications of such improvement when surgical treatment is undertaken. The third is whether or not a definite source of the bleeding can be demonstrated, such as an aneurysm or an angioma, and whether or not this source is operable. The fourth is the location of the hematoma; a clot in the temporal lobe, far frontal, or in the occipital lobe being far more favorable than one in the internal capsule and the basal ganglia. Fifth, and perhaps most important, is whether or not the patient has a history of widespread cardiovascular-renal arteriosclerotic disease and shows the characteristic signs thereof, with or without hypertension. In fact, hypertension may be a temporary concomitant of the "stroke." Given a patient with intracerebral hematoma, but without history or signs of cardiovascular-renal sclerosis; who has not lapsed into

From The Neuroclinic, Atlanta.
Submitted for publication April 24, 1954.

coma or whose state of consciousness is improving; who has a demonstrable aneurysm on the affected side; and whose clot is in the far frontal or temporal lobe, especially if on the right, this patient deserves operation even if he is over 60. It might be added, for those who think of intracerebral clot as almost always occurring in the internal capsule, that Grant and Austin[3] found from one-third to one-half in the temporal lobe.

REPORT OF CASES

CASE 1.—A 67-year-old white woman became mentally confused while at church. She was taken home, where she vomited for one week. Between the seventh and ninth days there was some improvement in her mental status and she was able to recognize her relatives, but she was unable to retain any food and had to be fed intravenously. She had difficulty in raising her left arm. Her past history was not suggestive of serious arteriosclerotic disease. Examination revealed a left homonymous hemianopsia and a left hemiparesis; examination of the fundi discovered no papilledema and no evidence of arteriosclerosis. Ventriculographic examination (Fig. 1) showed absence of the right temporal horn of the lateral ventricle. On craniotomy a large clot was aspirated from the temporal lobe. Two and one-half years later, except for a tremor of her head, neurologic examination gave negative results.

Comment.—This patient's age was overbalanced by the factors that she was never in coma, that she tended to improve, that her clot was in the temporal lobe and that she showed no evidence of generalized arteriosclerosis.

CASE 2.—A 47-year-old woman, known to be hypertensive, suffered a sudden onset of deep coma. She had complete right hemiplegia. The fundi showed no papilledema and no evidence of sclerotic vessels. The spinal fluid pressure was above 300 mm. of water, and angiographic investigation demonstrated a frontal intracerebral mass (*Cr*, Fig. 2). At craniotomy a sausage-shaped hematoma traversing the entire left frontal lobe from front to back was evacuated. The postoperative course was stormy, but two years later the patient was neurologically intact and managing her own business.

Fig. 1.—Ventriculogram showing absence of right temporal horn.

CASE 3.— A 52-year-old white man had a sudden onset of headache, aphasia and disorientation. Various diagnoses, including hysteria and meningitis, had been considered. An angiogram showed a space-taking lesion in the left temporal lobe and an aneurysm of the left middle cerebral artery. At craniotomy the clot was evacuated and the aneurysm clipped off. Ten months after the operation the patient was symptom-free and back at work.

CASE 4.— A 43-year-old woman had a sudden and severe onset of headache. She began to vomit, and fell, owing to weakness of the left arm and leg. She became disoriented. Her past history was free of symptoms of renal or cardiac disease, and she had never had hypertension. A ventriculogram revealed a space-taking lesion in the right temporal lobe, which proved to be an intracerebral hematoma and which was removed at craniotomy. Two months later she was much improved and able to come to the office. She still showed a mild weakness of the left side and left homonymous hemianopsia.

CASE 5.—A 62-year-old white man had a severe pain in the right side of his head and became temporarily unconscious. On his re-

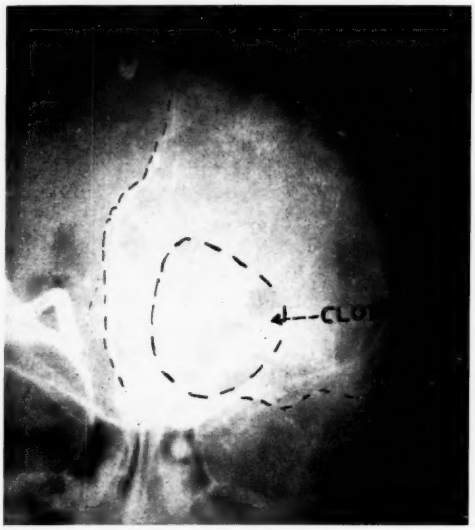


Fig. 2.—Frontal mass which proved to be a hematoma traversing the entire frontal lobe.

gaining consciousness, his family noticed that he had a right-sided facial weakness, slurred speech and difficulty in swallowing. This patient was known to be hypertensive, and cardiac disease was suspected. He had at times suffered from dynspnea and ankle edema.

Examination revealed left homonymous hemianopsia, a marked left hemiparesis, and partial aphasia. The fundi showed far advanced arteriosclerosis. The spinal fluid pressure was 290 mm. of water, and the fluid was bloody. A ventriculogram (Fig. 3) indicated a space-taking lesion in the temporal area. A left temporal clot was found and removed at craniotomy. One year later he had a serious recurrence.

Comment.—In addition to his age, this patient had unmistakable evidence of severe arteriosclerotic disease. This combination renders the subject unfavorable for operation.

CASE 6.—A 49-year-old white male had extreme hypertension of ten years' duration. He had lost 5 siblings in recent years from cerebrovascular accidents. In addition, his father and two maternal aunts had died from this cause. Two weeks before examination he had evidently become confused and had driven into the back of another car. Severe headache ensued. He improved for forty-eight hours, after which time there developed right hemiparesis and some speech disturbance. His state of consciousness from then on deteriorated steadily. When he was first seen by us he was deeply comatose, was hyperventilating and had a rattling type of respiration. Ophthalmoscopic examination revealed left papilledema and several small retinal hemorrhages, but no evidence of arteriosclerosis. The spinal fluid pressure was 470 mm. of water. While awaiting operation he went into decerebrate rigidity. A left temporal craniotomy was done, with removal of an intratemporal hematoma. When last seen, five months later, he was leading an active life and driving his own car.

SUMMARY

1. Age over 50, as a single factor, does not necessarily contraindicate operation for spontaneous intracerebral hematoma.

2. Hypertension, although extreme and of long standing, does not in itself constitute a contraindication.

3. Five other factors influence the decision for or against surgical intervention, namely, whether or not the patient is ever in coma; whether or not, if in coma, he tends to show improvement; whether or not a demonstrable cause of the hemor-

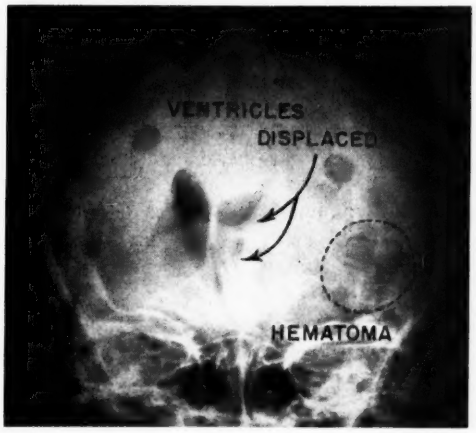


Fig. 3.—Ventriculogram showing a space-taking lesion in the temporal area, which proved to be a clot and was removed at craniotomy.

rhage, such as aneurysm or angioma, is present; whether or not the hematoma lies in a location favorable for surgical approach; and, most important, whether or not the history and the results of examination indicate the presence of generalized arteriosclerotic disease.

ZUSAMMENFASSUNG

1. Ein Lebensalter von mehr als fünfzig Jahren stellt als einzelner Faktor nicht unbedingt eine Kontraindikation der Operation eines spontanen Blutergusses im Gehirn dar.

2. Auch die Blutdruckserhöhung, selbst wenn sie extrem ist und lange besteht, bildet an und für sich keine Kontraindikation.

3. Die Beantwortung fünf anderer Fragen spielt bei der Entscheidung für oder gegen den chirurgischen Eingriff eine Rolle, nämlich

(a) ob der Patient im Koma ist,
(b) ob er, wenn im Koma, Neigung zur Besserung zeigt,
(c) ob sich eine Ursache der Blutung wie z. B. ein Aneurysma oder ein Angiom nachweisen lässt,
(d) ob die Lage des Hämatoms für einen chirurgischen Zugang günstig ist, und
(e) ob (sehr wichtig!) die Untersuchung das Bestehen einer allgemeinen Schlagaderverkalkung ergibt.

RESUMEN

1. La edad sobre 50 años como factor único no constituye necesariamente una contraindicación para la operación del hematoma intracraneal.

2. La hipertensión, aun intensa y de larga duración no constituye por si misma una contraindicación.

3. Los otros cinco factores que intervienen en la desición operatoria son, si el paciente esta ó no en coma, si esta en coma si tiende a mejorar, si existe una causa demostrable de hemorragia ó no, si existe un aneurisma ó angioma, si el hematoma esta situado ó no en un sitio favorable para el abordaje quirúrgico, y lo que es más importante si la historia ó la exploración física indica la existencia de arterioesclerosis generalizada.

RÉSUMÉ

1. L'âge (plus de 50 ans) ne doit pas être pris en considération comme seul facteur pour une contre-indication de l'opération de l'hématome intracérébral spontané.

2. L'hypretension, même extrême et d'une longue durée, ne constitue pas en elle-même une contre-indication.

3. Cinq autres facteurs influenceront l'indication ou la contre-indication opératoire : le patient est-il dans le coma ou non ? En cas de coma, présente-t-il une amélioration ou non ? présence ou absence d'un symptôme d'hémorragie, tel qu'un anévrisme ou un angiome; l'hématome est-il localisé à un endroit favorable pour l'intervention chirurgicale ? La présence d'une artériosclérose généralisée décelée par l'anamnèse et l'examen du malade est le facteur de plus important.

RIASSUNTO

1. Un'età di oltre 50 anni non controindica, di per se stessa, un intervento per ematoma intracerebrale spontaneo.

2. Uno stato ipertensivo, anche se molto accentuato e di lunga data, non rappresenta di per sè stesso una controindicazione.

3. Cinque altri fattori, viceversa, indicano o controindicano l'intervento. E sono: se il paziente è, o non è, ancora, in coma; se il coma tende, o meno, a regredire; se è dimostrabile, o meno, la causa dell'emorragia (come un aneurisma o un angioma); se la sede dell'ematoma è chirurgicamente raggiungibile, o meno; e, sopratutto, se l'anamnesi e l'esame obbiettivo indicano, o meno, la presenza di uno stato arteriosclerotico generalizzato.

REFERENCES

1. Aring, C. D.: Early Treatment of Stroke, Cincinnati J. Med. 30:304, 1949.
2. Odom, G. L.: Bloor, B. M., and Woodhall, B.: Intracerebral Hematomas in 106 Cases, Southern M. J. 45:936 (Oct.) 1952.
3. Grant, F. C., and Austin, G. M.: The Surgical Treatment of Spontaneous and Traumatic Intracerebral Hemorrhage, Am. J. M. Sc. 219: 237 (March) 1950.
4. Scott, M.: N. S. Treatment of Spontaneous Intracerebral Hemorrhage Simulating Common Stroke, Am. Int. Med. 37:751 (Oct.) 1952.
5. Browder, E. J., and Corradini, E. W.: Surgical Treatment of Intracerebral Hematoma, Arch. Neurol. & Psych. 65:112, 1951.
6. Falconer, M. A.: Future of Surgery in Treatment of Spontaneous Cerebral Hemorrhage, Lancet 2:945 (Nov. 15) 1952.
7. De Takats, G., and Graupner, G. W.: Emergency Treatment of Apoplexy, Practitioner 164: 242 (March) 1950.
8. Margolis, G.; Odom, G. L.; Woodhall, B., and Bloor, B. M.: Role of Small Angiomatous Malformations in the Production of Intracerebral Hematomas, J. Neurosurg. 8:564 (Nov.) 1951.

Some years ago I took part in one of those mass consultations which only the very rich or the very important are unfortunate enough to demand. I say this with no trace of cynicism, for when many experts meet together, either they agree, in which case the same verdict would have been reached more quickly and cheaply by one of them acting alone, or they disagree, and the line of action finally decided upon is not the clear-cut and definite decision of one mind, but a compromise representing the modicum of agreement, remaining after conflicting views have cancelled out. In this case the patient was an eminent scientist affected with severe diabetes, and also the victim of an enormous carbuncle, which had been excised. There were present besides myself, a pathologist, a bacteriologist, two titled physicians, another surgeon and the general practitioner. Discussion ranged around cardiac stimulants, vaccines, staphylococcal sera, injections of colloidal metals, intravenous antiseptics, immuno-transfusions and further surgery. (This was before the days of chemotherapy.) We had before us charts, and blood tests of every description. Meanwhile Lord Horder sat by himself studying the nurse's report book, and elucidated the clinical fact that was of far more importance than all these laboratory measures—that the intake of fluids had for some days been grossly insufficient, and that the urine output had fallen to 8 to 10 ozs. in the twenty-four hours.

—Ogilvie

Venous Angioma: A Cause of Cerebral Palsy*

SAMUEL ROSNER, M.D., F.I.C.S.

NEW YORK CITY, NEW YORK

IN a previous paper,[1] I reviewed the outstanding work of William Sharpe[2] on the pathologic aspects of cerebral birth palsy. He gave incontrovertible proof that about 9 per cent of newborn children show blood in the spinal fluid at birth. The cerebrospinal fluid was obtained by lumbar puncture of newborn children in the first twenty-four hours of life. He showed in this series of 500 lumbar punctures that the bloody cerebrospinal fluid in many cases was observed in children who had been delivered without instruments. It is Dr. Sharpe's contention,[2] with which I wholly agree, that cerebral birth palsy would be obviated in many cases if spinal punctures were carried out in all cases of suspected neonatal intracranial bleeding. In some cases of massive intracranial bleeding, craniotomy, with evacuation of the clot within the first few days, gave excellent results in Dr. Sharpe's hands.

The present paper deals with the surgical pathologic picture and the short-term postoperative results in a small series of cases. The patients selected for this demonstration showed the clinical picture of cerebral birth palsy. It is my opinion that the pathologic condition observed at operation was caused in every case by moderate intracranial hemorrhage at birth. In Case 2 it may have been due to meningitis.

In my experience, children who show the clinical picture of cerebral spastic paralysis are of two types. One type, by far the more numerous, shows adhesive arachnoiditis and arborization of abnormally large veins—usually one wide vein with many branches leading into the main trunk. The vessels are often accompanied by thin arteries and feed into a venous lake or aneurysm, which may invade the cortex up to 1 inch (2.5 cm.) or more in depth. These vessels are observed in the motor area just above the sylvian fissure and are usually in the dominant cerebral hemisphere. On the opposite cerebral hemisphere there is usually arachnoiditis, with a slight increase in the number of cortical veins. The brain, upon being exposed after the opening of the dura mater, is at first dusky, bulging and nonpulsating. Within ten to fifteen minutes the brain becomes pinkish and begins to pulsate normally. There is very little cerebrospinal fluid when the dura mater and arachnoid membranes and adhesions are incised. However, the flow usually becomes free shortly after the brain begins to pulsate.

When the abnormal cortal veins, which constitute what may be called a venous angioma, are cauterized with coagulating current, the brain recedes from the craniotomy opening and no longer shows the signs of abnormal intracerebral pressure. This happens within fifteen to twenty minutes after cauterization of the vessels.

In 1 of the 8 cases in this series there were signs of block in the fourth ventricle. This block was partial and was associated with hydrocephalus, as well as with spasticity of all limbs. The cause of the obstruction was adhesive arachnoiditis.

When the dura mater was exposed over the cerebellar lobes, the dura was bluish, bulging and nonpulsating. When the dura was incised in a stellate manner, the right cerebellar lobe was depressed and nonpulsating. There was a slow dribble of cerebrospinal fluid, which changed to a rush of fluid under pressure when the walls of the cyst were disrupted. The flow from the fourth ventricle then became free; the cerebellum began to pulsate normally, and the bluish color of the cerebellum changed to pink. The depressed cerebellar lobe be-

Submitted for publication April 15, 1954.

Most of the patients in the cases reported were treated at the Mother Cabrini Memorial Hospital.

gan to rise toward the craniotomy opening.

In 2 patients who died shortly after operation the cortex and white matter were of normal thickness despite the fact that 1 of the deceased patients had had moderately severe hydrocephalus as well as all the signs of cerebral palsy, involving all four limbs.

The clinical pathologic manifestations in all the cases consisted of motor aphasia and spastic quadriplegia. Intelligence could not be adequately tested because of the severity of the spasticity and the concomitant aphasia. The children ranged in age from 9½ months to 8 years.

REPORT OF CASES

CASE 1.—A. G. T., a boy 9½ months old, had undergone breech delivery. The diagnosis of intracranial hemorrhage was made a few days after birth. Spastic quadriplegia was present, more severe on the left side. Elevated intracranial pressure was noted on examination. The baby was mentally retarded. The operative pathologic picture consisted of right-sided venous angioma at the lower end of the motor area, involving the sylvian fissure and the undersurfaces of the temporal and frontal lobes. Postmortem examination showed the aforementioned pathologic changes, with an otherwise normal cortex and normal ventricles. The cause of death was medullary edema following operation.

CASE 2.—P. J. S., a boy aged 2½ years, had laryngeal spasm, hydrocephalus, spastic quadriplegia, mental retardation and motor aphasia, which came on gradually after the age of 11 months. Elevated intracranial pressure was observed on two occasions by spinal tap. At operation there was an adhesive arachnoiditis cyst blocking the fourth ventricle. At autopsy, dilated ventricles and remnants of adhesive basilar arachnoiditis were observed. The cerebral cortex and the white matter were within normal limits. The brain was heavier than normal. The cause of death was bronchopneumonia.

CASE 3.—D. R. S., a girl aged 8 years, had been injured at delivery. Bloody cerebrospinal fluid was observed on lumbar puncture a few days after birth. The patient was of the quadriplegic-clonic athetoid type. Clonic spasms involved the face as well as the limbs. The child had aphasia and laryngeal spasm, with difficulty in breathing and swallowing. Bilateral temporoparietal craniotomy revealed a left venous angioma in the lower portion of the motor area, with adhesive arachnoiditis. At the time of writing this child has improved enough to be admitted to a school for special training.

CASE 4.—G. K. Jr., a boy aged 2½ years, had cerebral palsy, diagnosed at a university medical center when he was 7 months old. Spastic quadriplegia, aphasia, epilepsy, amblyopia, mental retardation and elevation of the intracranial pressure were observed. The brain, viewed through a left frontoparietal craniotomy, showed adhesive arachnoiditis and venous angioma over the left motor area, just above the lower end of the sylvian fissure. At the time of writing the child's vision has improved. He recognizes his parents, laughs normally and eats well. He has had no convulsions since the immediate postoperative period and is no longer spastic.

CASE 5.—P. J. H., a girl 3 years old, was born prematurely. She was in the hospital for three months and in an incubator for eight weeks. She had spastic quadriplegia, motor aphasia, difficulty in breathing, mental retardation and signs of increased intracranial pressure. Surgical investigation through a left frontoparietal craniotomy revealed adhesive arachnoiditis, with a venous angioma in the lower portion of the motor area and involving the lower portion of the sylvian fissure. At the time of writing this patient is able to take steps with help, is not spastic and shows mental improvement.

CASE 6.—P. A. A., a girl aged 2 years, had cerebral spastic paralysis and was mentally defective and epileptic. At operation, angiomatous veins and adhesive arachnoiditis were observed in the left premotor area. The operative results are promising. The patient tries to walk and is no longer spastic. She had one epileptic attack after the operation but has had none since.

CASE 7.—A. W., a boy aged 6 years, had cerebral palsy, gross mental deficiency, epilepsy and an asymmetric skull. Operation revealed left adhesive arachnoiditis and venous angioma. The results of treatment are promising. One postoperative epileptic attack occurred while the patient had measles, but none have occurred since then. Mental and motor power are improving, and spasticity has greatly diminished.

CASE 8.—J. E. G., a girl aged 2½ years, was

mentally deficient and had cerebral palsy. Operation revealed a venous angioma in the left premotor area. The results of operative treatment are promising. The child is no longer spastic, and both mental and motor power show improvement.

COMMENT

Eight children with cerebral palsy of the spastic quadriplegic type were operated upon. Seven of the 8 patients could not sit up before the operation. The first patient of this series was operated on less than four months prior to the time of writing. In all cases adhesive arachnoiditis was present. In 1 case, hydrocephalus and the clinical picture of spastic quadriplegia were caused by an arachnoiditic cyst blocking the fourth ventricle. In the other 7 cases, adhesive arachnoiditis surrounding a venous angioma of the lower portion of the motor area was observed. The brain had a bluish cast. The pathologic changes were in the dominant hemisphere as a rule. In the child who underwent a bilateral craniotomy, adhesive arachnoiditis was present only on the opposite side. It is my opinion that only children with a moderate amount of intracranial bleeding live long enough for cerebral palsy to develop, and that nature tries to combat the bloody exudate by setting up an abnormal, localized system of cortical and subcortical veins which cause abnormal cerebral physiologic conditions through local congestion, edema, and hypoxia. When the bloody fluid is absorbed, arachnoiditic adhesions and abnormal veins are formed.

The surgical procedure is as follows:

1. An opening about the size of a half dollar is made over the lower end of the sylvian fissure. This is over the dominant lobe, usually. However, clinical observations, and diagnostic tests will help the surgeon to identify the side to be operated on. Both sides are treated if the brain does not pulsate normally, if normal color does not appear after electrocauterization of the vessels or if the brain shows signs of tension.

2. The branches of the middle meningeal vessels are cauterized, and the dura mater is opened in a stellate manner.

3. On incision of the dura mater, the abnormal vessels often bleed profusely. Nevertheless, they should be exposed and cauterized with the electrocautery. There is often a venous lake, which must be cauterized to its depth.

4. The dura mater is left open.

5. The temporal muscle is repaired with separate silk sutures. The fascia is left open.

6. The skin is repaired in one layer with separate silk sutures.

Before the operation a cutdown on a vein should be done if necessary, so that blood can be given promptly. Anesthesia is started with open drop ether and goes on to light intravenous sodium pentothal. Procaine hydrochloride is injected at the site of incision.

The promising results obtained far thus convince me that this procedure merits further trial.

SUMMARY

Eight cases of cerebral palsy are described. In 7 of the cases cerebral venous angioma, arachnoiditis and hypoxia of the brain were present. In 1 case there was an arachnoiditic cyst partially blocking the fourth ventricle and causing hydrocephalus and cerebral palsy.

Electrocauterization of the angioma resulted in better oxygenation and normal pulsation of the brain. Clinically, the 6 surviving patients showed definite improvement in spasticity and motor power. Mental status was improved in the mentally deficient children, and epilepsy was improved or totally eliminated in the epileptic children.

Author's note: Dr. Hyman Goldstein gave immeasurable help by his evaluation of the mental status in this series and by his posthospital pediatric care.

ZUSAMMENFASSUNG

Es werden acht Fälle von zerebraler Lähmung beschrieben. In sieben Fällen

handelte es sich um ein venöses Angiom mit Arachnoiditis und Sauerstoffverarmung des Gehirns. In einem Falle verlegte eine arachnoiditische Zyste teilweise den vierten Ventrikel und führte zu Hydrozephalus und zerebraler Lähmung.

Elektrokauterisierung des Angioms führte zur Verbesserung der Sauerstoffversorgung und zu normaler Pulsierung im Gehirn. Die sechs überlebenden Patienten zeigten klinisch ein deutliches Nachlassen der Spastizität und eine Verbesserung der motorischen Funktion.

RÉSUMÉ

L'auteur décrit huit cas de paralysie d'origine cérébrale. Sept de ces cas présentaient un angiome veineux cérébral, une arachnoïdite et une hypoxémie du cerveau. Dans un cas on trouva un kyste arachnoïdien bloquant partiellement le quatrième ventricule et causant de l'hydrocéphalie et de la paralysie d'origine cérébrale.

L'électro-coagulation de l'angiome provoqua une oxygénation meilleure et une pulsation normale du cerveau. Les six malades survivants montrèrent cliniquement une amélioration dans la spasticité et dans la motilité.

L'état cérébral fut amélioré chez des enfants mentalement déficients, et chez des enfants épileptiques on constata une disparition partielle ou totale des crises épileptiques.

RIASSUNTO

Vengono descritti 8 casi di paralisi muscolare cerebrale. In 7 di essi vi era un angioma venoso cerebrale, con aracnoidite e ipossia del cervello. In 1 caso vi era un'aracnoidite cistica che bloccava parzialmente il IV ventricolor ed era causa di idrocefalo e paralisi.

L'elettrocoagulazione dell'angioma condusse ad una miglior ossigenazione del cervello e alla comparsa di pulsazioni normali. Clinicamente i 6 pazienti che sopravvissero presentarono un netto miglioramento dello stato spastico e della forza.

RESUMEN

Se describen ocho casos de parálisis cerebral. En siete de ellos se encontraron angioma venoso, aracnoiditis e hipoxia cerebral. En uno se encontró un quiste aracnoideo bloqueando parcialmente al cuarto ventrículo y produciendo hidrocefalia y parálisis cerebral.

La electrocauterización del angioma produjo una mejor oxigenación y pulsatilidad normal en el cerebro. Clínicamente, los 6 pacientes supervivientes mostraron una mejoría definida en la espasticidad y poder motor.

REFERENCES

1. Rosner, S.: Neonatal Bleeding and Cerebral Birth Palsy, J. Internat. Coll. Surgeons **19**:494, 1953.

2. Sharpe, and Sharpe, N.: Neurosurgery: Principles, Diagnosis and Treatment. Philadelphia: J. B. Lippincott Company, 1928, pp. 100-146.

3. Goldstein, H.: A Study of Mongolism and Non-Mongolid Mental Retardation in Children, Arch. Pediat. **71**:11, 1954. Treatment of Mongolism and Non-Mongolid Mental Retardation in Children, to be published (Personal Communication).

4. Denis: Commercy, 1826.

5. Billard: Paris, 1828.

6. Cruveilhier :Paris, 1829-35.

7. Little, J. W.: Deformities of the Human Frame, Lancet **2**:350, 1843. On the Influence of Abnormal Parturition, Difficult Labors, Etc., Upon the Mental and Physical Condition of the Child, Obst. Tr. **3**:243, 1862.

8. MacNutt, S. J.: Am. J. Obst. **18**, 1885.

9. Beneke: Munchen med. Wchnschr. p. 2125, 1910.

10. Pott: Ztschr. f. Geburtsch u. Gynakol. **69**, 1911.

11. Mayer, A.: Zentralbl. f. Gynakol. **46**, 1915.

12. Halland: Tr. Edinburgh Obst. Soc., 1918, p. 40.

13. Schafer, Z.: Ztschr. f. Geburtsch u. Gynak. **64**, 1921.

14. Schwartz, R.: Munchen med. Wchnschr. **30**, 1922; Deutsche med. Wchnschr. Oct. 3, 1924.

15. Saenger, H.: Monatschr. f. Begurtsch u. Gynak. **65**:257-274, 1924.

16. Capon, N. B.: J. Obst. & Gynec. Brit. Empire **39**:572, 1922; Lancet **2**:1203-1206, 1924.

17. Barnett, E. J.: Northwest Med. **21**:243, 1922.

18. Fischer, B.: Schwarze med. Wchnschr., 1924.

19. Huenekens, E. J.: Care of the Newborn J.A.M.A. **81**:624, 1923.

Cerebral Venous Angioma Treated by Electrocautery

Report of a Case

SAMUEL ROSNER, M.D., F.I.C.S.

NEW YORK CITY, NEW YORK

CEREBRAL venous angiomas are essentially of three types: (a) the simple varix, (b) the serpentine varix and (c) the racemose or cirsoid varix.

They are usually associated with nevi of the face or body. These varicosities may cause changes in the retinal veins, which become tortuous. In some cases buphthalmos may occur, with cerebral venous angioma. This is known as the Sturge-Webber syndrome. The buphthalmos is associated with cerebral venous angioma on the same side.

The cerebral venous angioma is not a neoplasm in the strictest sense of the word, yet it causes signs and symptoms compatible with cerebral neoplasm. Headaches, personality changes, and epileptic attacks are often associated with cerebral venous angioma.

The patient in many cases of cerebral angioma is referred to the radiologist for high voltage roentgen treatment. This type of treatment is often successful. My one objection is that sometimes cerebral necrosis occurs in the area of irradiation. In my opinion, therefore, this method of attack is to be used only when surgical repair of the condition is not feasible.

The following case is interesting because of the objective observations and the response to treatment.

E. G., a right-handed man aged 50, was admitted to the hospital on Aug. 14, 1952. He was brought into the hospital because of a generalized convulsion that had occurred at home.

While in the hospital, the patient had three generalized attacks. During each attack his eyes would turn to the right; there was vomiting, but no localizing signs were observed except the ocular rotation to the right side.

The history of past illnesses was noncontributory. A close relative volunteered the information that there had been signs indicative of some personality changes for two years prior to the epileptic episodes; whereas the patient had been an extremely kind, considerate person all his life, during the past two years he had become very irritable over little things. He had showed loss of memory and had indulged in alcoholic beverages frequently.

During the examination the patient was cooperative but confused, and cerebration was dull. He had been given anti-epileptic medication prior to the examination.

Neurologic Observations.—There was mild weakness of the central type in the seventh and twelfth nerves on the right side. Abdominal reflexes were absent, and bilateral positive Babinsky reactions were noted. In all other respects the results of neurologic examination were within normal limits.

Pneumoencephalographic investigation was carried out by means of lumbar puncture. The cerebrospinal fluid was clear and under pressure. The Pandy test gave a positive result.

The roentgenograms showed the lateral ventricles to be dilated. The right lateral ventricle was not distorted, but the left ventricle showed the occipitotemporal horns to be pushed toward the midline, with partial obliteration of the posterior pole; the left temporal horn was almost completely obliterated.

It was my opinion that the history, signs and symptoms were compatible with cerebral neoplasm in the left temporo-occipital region. There was one interesting observation: definite atrophy of the brain, as shown by pneumoencephalographic examination, was present.

A few days after the pneumoencephalographic procedure the patient was reexamined.

Submitted for publication Feb. 10, 1954.

There was Rombergism to the right, the patient walked on a wide base and showed left homonymous quadrantic loss of visual fields. This was determined by confrontation test.

On Aug. 23, 1952, the patient underwent left temporoparieto-occipital craniotomy.

Craniotomy.—A left-sided craniotomy was performed. A 3-inch (7.5 cm.) linear incision was made from the temporal region, running toward the left parietal eminence. The skull showed bluish discoloration in the area of incision. The bone was vascular. An opening in the skull somewhat larger than a silver dollar was made. This was centered at the upper end of the fissure of Sylvius. The dura showed dark blue discoloration in this area. The dura was incised in a stellate manner. Four large veins, almost 1 cm. in diameter, converged in a central lake at the upper angle of the fissure of Sylvius. Each vein was cauterized from its extremity inward toward the confluence of veins. Each vein in turn bled profusely when the cautery tip entered its bed. It was possible, in each case, however, to pick up the vein with a hemostat and complete the coagulation within a few seconds.

The incision was closed with temporal muscle and skin. The patient was sent back to bed in good condition. He was given 1 pint of blood postoperatively and reacted well thereafter.

Postoperative Course. — Cerebration was greatly improved. Insight into the problems the patient would have to face was good. Walking and balance improved. Speech was rational and had improved in clearness. There were no postoperative epileptic attacks in the hospital, and the patient was discharged on the eighth postoperative day. After a protracted convalescence, the patient at the time of writing, one and one-half years after the operation, is completely free from epileptic attacks. He is talking Dilantin (gr. 3) daily. Cerebration is improved and adequate, and there has been no further deterioration in behavior.

SUMMARY AND CONCLUSIONS

The author reports a case of cerebral venous angioma in a man aged 50, treated by electrocautery.

This type of patient is usually treated by high voltage roentgen therapy, but, since cerebral necrosis sometimes is associated with this type of treatment, he prefers direct attack when possible.

A point of interest is the fact that the pneumoencephalogram was indicative of a space-occupying lesion. This would indicate that venous angiomas cause local cerebral edema.

Also significant is the fact that the complete obliteration of veins leading into the point of confluence of the varices did not cause any cerebral upset or loss of function, but rather resulted in almost immediate improvement. This would tend to prove that another means of venous drainage came into function almost immediately.

The final point is the rapid postoperative improvement observed in the neurologic and psychic status of the patient, despite the fact that there was definite brain atrophy, as shown in the pneumoencephalogram. The history of alcoholism was also a significant point with regard to the brain atrophy and the postoperative improvement.

RÉSUMÉ ET CONCLUSIONS

L'auteur décrit un cas d'angiome veineux cérébral chez un homme âgé de 50 ans, traité par électro-coagulation.

ZUSAMMENFASSUNG

Der Verfasser berichtet über einen mit Elektrokauterisierung behandelten Fall eines zerebralen venösen Angioms bei einem fünfzigjährigen Manne.

RESUMEN Y CONCLUSIONES

El autor comunica un caso de angioma cerebral venoso en un hombre de 50 años de edad tratado por electrocauterio.

CONCLUSIONI RIASSUNTIVE

L'autore riporta un caso di angioma venoso cerebrale in un uomo di 50 anni trattato con elettro-cauterio.

Diastematomyelia

EDWARD MARTIN GATES, M.D., F.A.C.S., F.I.C.S.,
AND JAMES A. MORTON, M.D., F.A.C.R.
PONTIAC, MICHIGAN

DIASTEMATOMYELIA is a developmental malformation in which the spinal cord or cauda equina is divided into two parts. Duplication is generally effected by the presence of a midline division of the spinal canal by a boney or fibrocartilagenous septum arising from the posterior surface of the vertebral body and passing dorsal through the spinal canal. Herren and Edwards[1] noted the coexistence of spina bifida or spina bifida occulta in about half of the cases. These authors reviewed the literature and collected 42 cases, to which they added one of their own. Pickles[2] and Neuhauser, Wittenberg and Dehlinger[3] have subsequently reviewed the literature and added cases of their own. To these we wish to add the following case.

REPORT OF CASE

S. S. is a 4-year-old girl whose mother brought her for medical consultation on April 25, 1951. The mother stated that since the child's birth there had been a large mane of dark brown hair hanging from her back. The child developed normally and learned to walk at the age of 1. Six months prior to consultation the child began to complain of pain in the back, which was persistent but did not radiate. The mother stated that at the same time the child began to complain of pain, her gait showed a change to a clumsy waddling walk.

The remainder of the general and neurologic history was noncontributory. General physical examination showed the child to be entirely normal as to growth and development, except for the presence of a heavy growth of hair (Fig. 1) arising in the back, in the region of the lower thoracic and the upper lumbar area. The hair could be combed out to hang as far down as the patient's gluteal folds. Neurologically the child was normal except for a gait midway between a waddle and a spastic type of movement of the legs. Bilaterally the quadriceps femoris reflexes were markedly increased, as were the Achilles reflexes. There was a bilateral Babinski reflex. Sensory examination, so far as could be determined in a child 4 years old, revealed no abnormalities. The anal sphincter reflex was normal. The results of laboratory studies were within normal limits. The protein content of the spinal fluid was 18 mg. per hundred cubic centimeters. Roentgenograms of the spine were taken, and Dr. Morton reported them as showing a spina bifida defect of the twelfth thoracic segment, with supernumerary ossification centers involving the first and second lumbar vertebrae, producing widening of the bodies. The pedicle shadows were spread apart, but were normally rounded and showed no erosion or flattening (Fig. 2).

A pantopaque myelogram was then taken, which showed the pantopaque column splitting into two columns at the upper edge of the second lumbar body (Fig. 3) and reuniting into a single column at the level of the eleventh thoracic vertebrae. The outlined oval appeared to have a central osseous mass or spicule arising from the upper part of the first lumbar body. The diagnosis of diastematomyelia was made, and operation was performed on April

Fig. 1.—Hairy malformation in lumbothoracic area of patient with diastematomyelia.

Submitted for publication Oct. 2, 1953.

30. An incision was made from the level of the eleventh thoracic spinous process to that of the second lumbar spinous process, and the muscles were reflected bilaterally from the spinous processes and the respective laminae. A bilateral laminectomy was begun at the level of the second lumbar lamina and was extended upward. At the first lumbar level, no spinous process was found, and the laminae were united in the midline by a fibrocartelagenous bridge. As the laminectomy was continued at the first lumbar level, a midline osseous spur was observed, arising from the fibrocartilaginous band and extending through the dura to the body of the first lumbar vertebra. The dura was divided at this level into two complete sacs, which united into a common sac at the level of the eleventh thoracic vertebra. The cauda equina was divided.

A division of the lower sacral segements of the cord could not be detected. The bony portion was rongeured away, and its wide base of orgin on the first lumbar body was waxed in an effort to prevent possible regeneration of the septum. The operation was ended with the usual closure. The pathologic report on the specimen was that of a small osteoma showing no evidence of malignancy. The patient's postoperative course was uneventful, and she was discharged home on the tenth postoperative day. Examination on final discharge, three weeks after the operation, showed a return of growth of the large mane of hair, normal quadriceps and achilles reflexes, and no evidence of a Babinski reflex in either foot. The gait was considered normal.

Fig. 2.—Anterior posterior roentgenogram of spine, showing a centrally placed bony spur at the level of the first lumbar vertebra, with widening of the spinal canal.

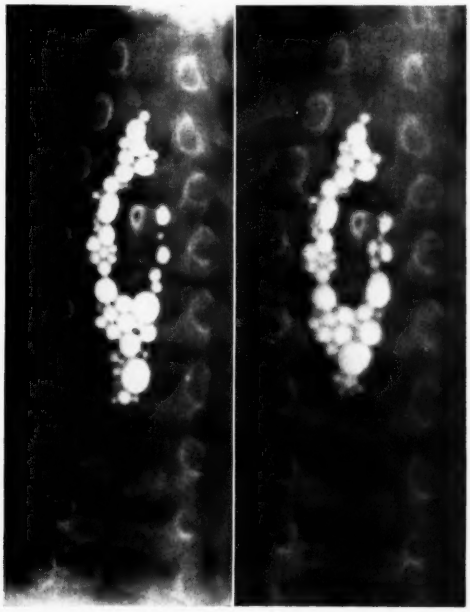


Fig. 3. — Myelogram showing the Pantopaque column splitting into two columns at upper edge of second lumbar vertebral body and reuniting at the level of the eleventh thoracic vertebra.

COMMENT

One may suspect the presence of diastematomyelia, a rare congenital malformation, by the presence of the hairy growth over the spine. It is well to realize, however, that a hairy growth in the vertebral area is not always associated with this defect, for in our last 4 cases, in which an equally dramatic amount of hair was

present, roentgenograms and myelograms were normal. When the roentgenogram shows a centrally placed area of ossification that appears as a spur arising from the body of a vertebra in the lateral films, the diagnosis of diastematomyelia can generally be made. Confirmation is obvious from the manner in which the contrast medium in the myelogram splits to pass around the defect, thus outlining a duplication of the thecal canal.

Surgical removal of the bony septum is indicated when neurologic symptoms are present and as a precautionary measure to prevent the development of neurologic disturbances resulting from hindrance of the normal ascent of the cord with growth. Such a centrally placed spur of bone could easily anchor the cord or the cauda equina, and result in the so-called Arnold-Chiari malformation.

CONCLUSION

A case of diastematomyelia is reported with roentgenograms showing the central defect and a myelogram showing duplication of the thecal canal. Laminectomy was performed, with removal of the bony septum, and was followed by relief of symptoms.

RÉSUMÉ

L'auteur présente un cas de diastématomyélie, avec un radiogramme montrant l'affection centrale et un myélogramme révélant un double canal thecal. On pratiqua une laminectomie avec ablation du septum osseux. Cette intervention fut suivie de régression des symptômes.

ZUSAMMENFASSUNG

Es wird ein Fall von Diastematomyelie vorgestellt. Die Röntgenaufnahmen zeigen den zentralen Defekt, und ein Myelogramm lässt die Verdoppelung des Durakanals erkennen. Es wurde eine Laminektomie mit Entfernung des knöchernen Septums ausgeführt. Danach verschwanden die Symptome.

RESUMEN

Se comunica un caso de diastematomielia con roentgenogramas mostrando el defecto central y un mielograma mostrando la duplicación del conducto tecal. Se realizó laminectomía con extirpación del septum óseo. Se obtuvo el alivio sintomático.

RIASSUNTO

Viene descritto un caso di diastematomielia, documentato da radiogrammi e da mielogrammi. Fu eseguita una laminectomia che consentì di asportare il setto osseo. Ne seguì la guarigione.

REFERENCES

1. Herren, R. Y., and Edwards, J. E.: Diplomyelia (Duplication of the Spinal Cord). Arch. Path. **30**:1203-1214 (Dec.) 1940.

2. Pickles, W.: Duplication of the Spinal Cord (Diplomyelia). J. Neurosurg. **6**:324-331 (July) 1949.

3. Neuhauser, E. B. D.; Wittenborg, M. H., and Dehlinger, K.: Diastematomyelia (Transfixation of the Cord or Cauda-Equina with Congenital Anomalies of the Spine), Radiology **54**:659-664 (May) 1950.

Pay no attention to what critics say. There has never been set up a statue in honor of a critic.

—*Sibelius*

Editorial

Section on Neurosurgery
A New Field of Cooperative Endeavor

IN this issue of the Journal we present, with great pleasure and satisfaction, the presentations, discussions and scientific aspects of the initial meeting of the Neurosurgical Section inaugurated at the Eighteenth Annual Congress of the United States and Canadian Sections of the International College of Surgeons in New York on Sept. 13, 1953. We feel that this new Section, in addition to meeting an urgent contemporary need, will become a valuable link in the coordination of neurosurgery with specialization in many allied fields. Every such cooperative effort, when sincerely conceived, efficiently planned and carried out in accordance with the ideals that originally led to its establishment, must reduce the undeniable dangers that lie in overspecialization and at the same time provide all surgeons with a central source of the information they need in correlating their own work with that of the profession in general. The International College of Surgeons is a many-branched organization, but it is an integrated one; and, as constant advances in research, discovery and technic place upon us all the obligation of acquiring as much knowledge and understanding as possible, we recognize the equal obligation of sharing both with our colleagues.

The first meeting of the Neurosurgical Section more than fulfilled our hopes and expectations. A full day's program was devoted to the spine and spinal column, beginning with studies on the physiologic mechanisms of spinal shock, spinal cord reflexes and regeneration of the cord and proceeding, later in the morning session, to surgical treatment of congenital spinal disorders and spinal cord tumors and cysts. The afternoon was given over largely to the management of injuries to the cervical portion of the spine, disturbances of the bladder from the neurologic point of view and the spasticity of paraplegia. The speakers and discussants were eminent men, authorities in their particular fields. As was fitting for an international group, several came to us from other countries, including Hawaii, Turkey and Sweden. Dr. Harold C. Voris of Chicago, who introduced the newly organized Section at the afternoon session, made the following remarks:

"This is the Neurosurgical Section of the International College of Surgeons in the United States and Canada, at whose Eighteenth Annual Congress we are now assembled. Quite a formidable title! But it must be pointed out that, since no part of the College excludes or is excluded from the others, and the Neurosurgical Section is an integral part of the international group, we find it highly gratifying to have had Dr. Hagbarth of Sweden on our morning program and now have the privilege of introducing Dr. Feyyez Berkay, Assistant Professor of Surgery and Neurosurgery at the University of Istanbul, who is a Fellow of this organization and of the American College of Surgeons and is certified by the American Board of Surgery."

Both Dr. Hagbarth and Dr. Berkay made valuable and significant contributions to the program, as did the distinguished American surgeons who participated. A panel discussion on the surgical treatment of the intervertebral disc from two points of view, that of the neurosur-

geon and that of the orthopedic surgeon, was both excellent and stimulating. At the forthcoming Nineteenth Annual Congress in Chicago in September, the idea will be carried on with a panel discussion of the painful neck, shoulder and arm.

The symposium type of program is admirably suited to our purpose, serving as it does the College ideal of organization for the study and correlation of knowledge and instruction in surgery and the sciences allied thereto. At the Nineteenth Congress our subject of discussion will be cancer of the brain. Not only primary and metastatic carcinoma but malignant glioma will be considered. There will be papers on angiography and the surgical management, as well as on the cyclotron and on radioactive isotopes and other chemical substances. The discussions will occupy a place on the program of an importance equal to that of the original presentations.

This brief account of the founding, first meeting and proposed activities of the Nurosurgical Section would be incomplete without an expression of thanks from myself as Chairman, Dr. Voris as Co-Chairman and Dr. Kenneth H. Abbott, Secretary, on behalf of the neurosurgical members of the College, to Dr. Max Thorek, Founder and Secretary General of the International College and Editor of the Journal, for his support in forming the new Section and his cooperation in publishing this special issue. With the Section as a rallying point, we believe that more neurosurgeons will wish to participate in our future programs. They will be cordially welcomed. We wish also to thank once more the authors and discussants who made our first Section meeting so memorable and revivifying an experience.

JAMES WINSTON WATTS, M.D.,
F.A.C.S., F.I.C.S., Chairman

Vesalius may be rightly described as the Father of the Renaissance in medicine, and his work was followed by William Harvey's discovery of the circulation of the blood. This knowledge is now the property of every schoolboy, but it is only three hundred years old, and it is interesting to reflect that this bit of information, which a lad of ten recognizes as a fundamental part of life, was not known to the great thinkers of the ages, including Plato, Aristotle, Cicero, Thomas Aquinas, St. Augustine, or the Apostle Paul. Vesalius and Harvey were followed by a host of anatomists who spent the next fifty years showing, among other things, how many mistakes in anatomy old Galen and his followers had taught for more than a thousand years.

—*Major*

New Books

Neurosurgery of Infancy and Childhood. By Franc D. Ingraham and Donald D. Matson. Springfield, Ill.: Charles C Thomas, Publisher, 1954.

Children are not miniature adults. A number of general surgeons who have had the opportunity of working in large children's hospitals have recognized this and have so perfected themselves in the surgical problems of childhood that they can be considered pediatric surgeons. However, the neurosurgical problems of children, like those of adult life, require a basic knowledge of the form, functions and disorders of the nervous system if they are to be properly dealt with. The authors of this book are well-known surgeons who have had extensive experience in adult neurosurgery. This book is based on their wide experience with neurosurgical problems in children at the Children's Medical Center in Boston. The extent of this experience is indicated by their treatment of over 1,400 children with spina bifida, and their series of 313 histologically verified intracranial tumors in children under 12 years of age, their 63 cases of intraspinal tumor in the same age group.

Other authors have taken up specific neurosurgical problems of childhood in books or monographs, especially the intracranial tumors, and a general survey of pediatric neurosurgery has been briefly attempted in a few articles. This is the first textbook of pediatric neurosurgery. It includes congenital anomalies of the central nervous system and its coverings; hydrocephalus; trauma; intracranial tumors; intraspinal tumors; infection; cerebral vascular disorders; epilepsy; encephalopathy, and finally pediatric neurosurgical anesthesia. The sections on congenital anomalies and hydrocephalus are the most original and represent the very important personal experience of the authors in this field.

The book is well illustrated with 380 illustrations and 1 plate, and the bibliography includes 206 references. Every neurologic surgeon and everyone concerned in the care of children should read this book and have it available for reference.

HAROLD C. VORIS, M.D.

Clinical Orthopaedics. Edited by Anthony F. DePalma. Philadelphia: J. B. Lippincott Company, 1954.

This is the third volume of a new series entitled *Clinical Orthopaedics*. It consists of 23 original articles written by well-known orthopedic surgeons and assembled by Anthony F. DePalma, the Editor-in-Chief of the series. This third number is divided into two sections. Section 1 consists of articles describing injuries or diseases of the soft tissue about joints and the methods of treating these conditions. A partial list of the subjects includes injuries to ligaments of the knee or ankle, cysts of and injuries to the semilunar cartilages of the knee, dislocations of the patella, and injuries to the rotator cuff of the shoulder. Section 2 includes articles of a more general type. A paper on giant cell tumors is particularly noteworthy. The first two volumes of *Clinical Orthopaedics* were accepted by the medical profession with considerable enthusiasm. This third volume have equal value for the orthopedic surgeon, as well as for all surgeons who are called upon to treat injuries to the spine and extremities.

EDWARD L. COMPERE, M.D.

The Chest. A Handbook of Roentgen Diagnosis. By Leo G. Rigler. Chicago: The Year Book Publishers, Inc., 1954. 2d ed. Pp. 380, with 338 illustrations.

It is surprising what a wealth of diagnostic material has been included in this small, very useful reference volume. The illustrations are excellent, well chosen and handy to the pertinent material in the text. Included are details of the examination technic and bases for interpretation of the roentgenograms. Stress is laid upon the clinical aspects of the work of the radiologist. The material from which the illustrations have been selected is well known for its extent and diversity. In his radiologic work the author exemplifies the ideal roentgen diagnostician and sets a high standard of proficiency for all who study his teachings.

JAMES T. CASE, M.D.

APPLICATION FOR HOTEL RESERVATIONS

Nineteenth Annual Congress
of the
INTERNATIONAL COLLEGE OF SURGEONS
United States and Canadian Sections

PALMER HOUSE

Chicago, Illinois — September 7 thru 10, 1954

Note: The headquarters hotel is reserving a sufficiently large block of rooms for those who will be attending the Congress. However, early reservations are necessary to hold this block of rooms.

PALMER HOUSE

Single Rooms	Double Rooms	Twin Rooms	Parlor and 1 Bedroom	Parlor and 2 Bedrooms
$7.50 and up	$12.50 and up	$12.50 and up	$30.50 and up (1 person)	$44.00 and up (2 persons)

Mail to: ROOM RESERVATIONS
THE PALMER HOUSE
CHICAGO 90, ILLINOIS

Please make the following reservation for the 19th Congress of the International College of Surgeons:

.....Single Room	.....Double Rooms	.....Twin Rooms	Parlor and .....1 Bedroom	Parlor and .....2 Bedrooms
$..... to $.....	$..... to $.....	$..... to $.....	$..... to $.....	$..... to $.....

Arriving.....a.m. or p.m. Departing.....a.m. or p.m.

Please include the names of all persons who will occupy rooms requested

.....

.....

Confirm reservation to:.....

.....

.....